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MINNESOTA MEDICINE

Minnesota State Medical Association

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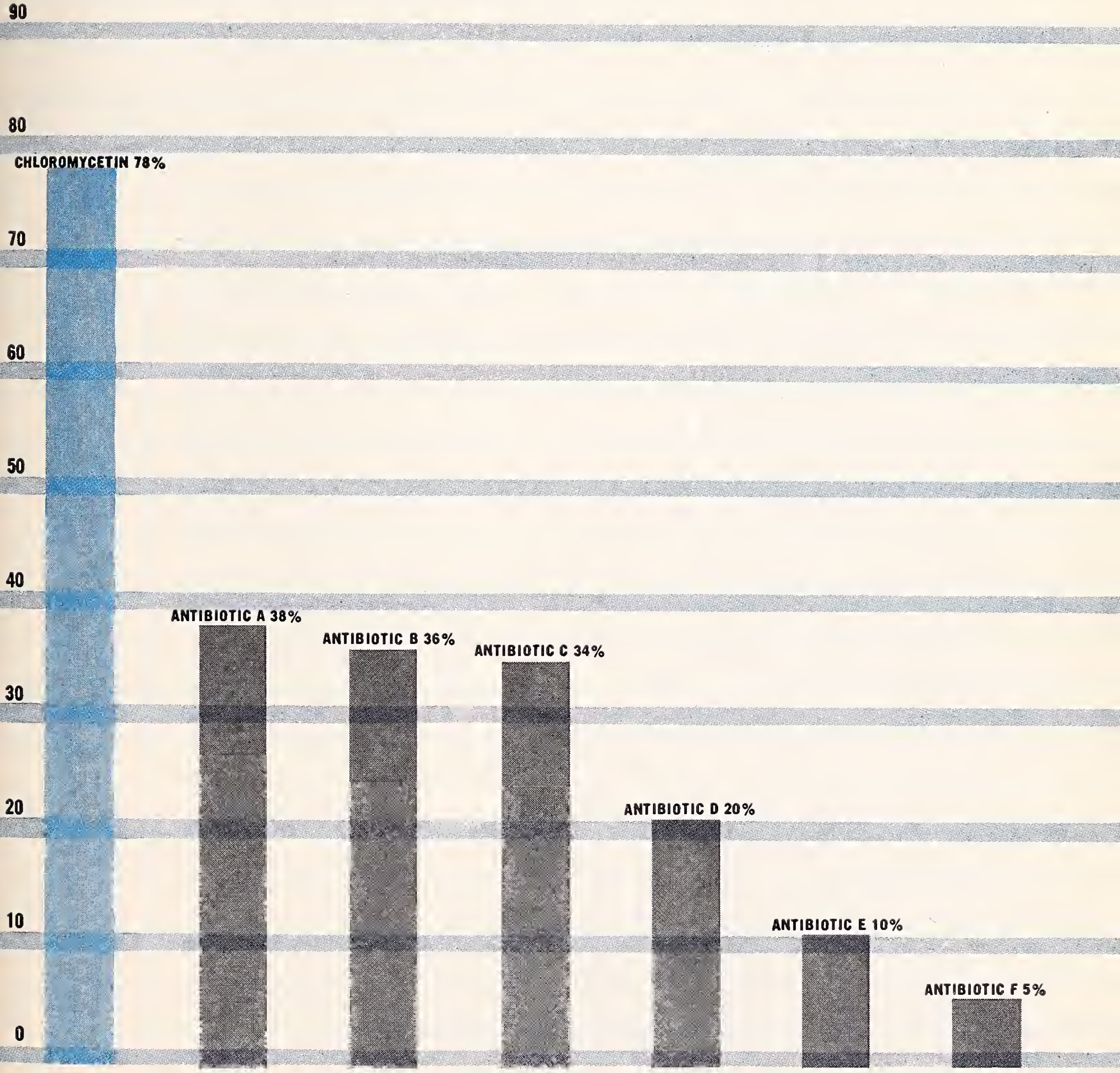
COMPARATIVE SENSITIVITY OF MIXED PROTEUS SPECIES TO CHLOROMYCETIN AND SIX OTHER WIDELY USED ANTIBIOTIC AGENTS*



*This graph is adapted from Waisbren and Strelitzer.[15] It represents *in vitro* data obtained with clinical material isolated between the years 1951 and 1956. Inhibitory concentrations, ranging from 3 to 25 mcg. per ml., were selected on the basis of usual clinical sensitivity.

Minnesota

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Original Contributions

Menopause and Thereafter

W. H. MASTERS, M.D.
St. Louis, Missouri

THE ONSET of the climacteric normally varies over a fifteen-year period for each individual woman. Thus, the first symptoms associated with the menopause frequently occur prior to the actual cessation of the menstrual flow. Efforts to alleviate the innumerable complaints associated with this female aging milestone have varied from complete therapeutic nihilism to extensive psychoanalytical considerations, There is, at present, no professional agreement as to an effective solution for this vexing problem of "the change of life."

It is inevitable that the more dramatic symptomotology of the menopause has occupied medical attention to the exclusion of significant consideration of future replacement measures necessitated by this dramatic definition of endocrine imbalance. As physicians, we must face the physiological fact that the postmenopausal years are, for the woman, a socially conditioned phenomenon. She, of all female animals, lives beyond her normal period of reproductivity. Since medical science has been directly responsible for the extra two decades of human life expectancy, the profession has the responsibility of giving adequate physical and mental support for the aging individual.

It has long been obvious that the major phases of our physiological life are growth, mature function, and involution. The influence of the glands of internal secretion upon the phases of growth and mature function have been well defined. It is important that we evaluate the entire endocrine system during the involutionary phase of life, if we are to define, effectively, the retrogressive process of aging.

Present knowledge of pituitary function during the postmenopausal years has been accumulated during the last ten years, as the result of detailed laboratory investigation. Albert[1] has supported data from our own laboratory[2] in the contention that the gonadotrophic secretory activity of the pituitary is not depressed by the aging process. In fact, there is a marked elevation of gonadotrophin production from the fiftieth to the seventieth year. Ultimately, of course, there is a reduction of secretory function to a new base level which is roughly half that of the mature function years. There is no question, however, of the continued gonadothrophin production by the pituitary, even into the eighty-year age group. Adrenocorticotrophin production has been described by Pincus[3] in a recent publication. It is obvious that the pituitary-adrenal axis continues to function with relatively unabated effectiveness during the involutionary phase of life.

As the result of the maintained equilibrium between the pituitary and adrenal glands, the adrenal is the most active secretory gland of the entire endocrine chain, subsequent to the menopause. Corticosteroid hormones are produced with relatively unabated effectiveness, even to the point of actually creating a catabolic imbalance in such basic physiological functions as protein balance and calcium storage. This is a prime example of independent glandular secretory activity once the axis of interglandular stimulation is destroyed.

The effectiveness of thyroid function during the aging process attracted the interest of investigators as early as 1917.[4] The first definition of thyroid influence during the aging process described at most a 12 per cent reduction in secretory effectiveness in the aging individual,[5] when compared to the twenty to forty-year age groups. Starr[6] has done definitive protein-bound iodine studies on a controlled aging population. He found that the levels obtained are roughly equal to those established for the mature function age groups. It is evident that a good deal of work still remains to be done before definitive statements can be made in terms of effective thyroid secretory activity during the aging process. It is equally obvious that all of the present evidence

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points toward minimal reduction in thyroid secretory activity. This is probably a reduction in physiological demand, rather than an innate inability of the gland to provide effective secretory potential.

Knowledge of the function potential of the pancreas dates from 1940, and the initial efforts of Meyer[7] and his associates. They described an effectiveness of secretory activity that is roughly 20 per cent reduced over what one might expect for the twenty to forty-year age groups. However, they also point out that this reduction in function would certainly be counterbalanced by the basic reserve potential of the pancreas, and that any demonstrable reduction in secretory effectiveness is, in reality, a theoretical rather than an actual physiological distress. Thus, barring established diabetic states, it is obvious that the pancreas also has the individual ability to return to the mature function status, given the proper exogenous stimulation. It should be noted in passing that the senile onset of a previously undefined diabetic state (even resistant to a prior pregnancy syndrome) is known to be associated with the sex steroid withdrawal of the menopause. The exact connection of the mutually stimulative mechanism between pancreas and an adequately functioning gonad has not been developed to date.

Gonadal secretory activity during the postmenopausal era is obviously at striking variance with the remainder of the endocrine chain. Certainly the menopause represents a major milestone in the involutional retrogression of the gonadal function. It is equally certain that there is evidence of continued ovarian secretory activity during the postmenopausal era.[8-10] However, this glandular activity is inadequate for the effective clinical support of many of the basic physiological processes of body economy. There is indisputable evidence of continued pituitary gonadotrophin production during the two decades after normal cessation of menstrual flow.[1,2] Since the ovary is apparently incapable of responding to normally stimulative channels, it must be presumed that permanent menopausal changes are in the direction of resistance to any basic stimulative mechanism. This premature resistance to physiological stimulation is the key to the aging process within the ovary.

It is important to re-emphasize the dual physiological role of the ovary during the mature function phase of life. The primary function of the ovary is obviously that of reproduction. The secondary function, infinitely more necessary to basic body economy, is that of sex steroid production. We normally tend to think of the climacteric as signifying the end of the female reproductive function. While certainly true, this functional involution is of minor import compared to the distress associated with the involution of clinically effective sex steroid secretory activity. If only reproductive activity failed with the menopause, and clinically effective steroid productivity was maintained, there would be no definition of the "neutral gender."[11] This reduction in steroid secretory activity by the gonad is the "Achilles' heel" of the entire endocrine system during the involutional phase of life.

As the result of the inability of the ovary to respond to usual gonadotrophin stimulation, the pituitary-gonadal axis is completely lost. Subsequently, the remaining members of the endocrine chain become uncoordinated secretory organs whose functional involution results from lack of clinical demand, rather than lack of secretory function reserve. Thus, we have the basic definition of the third sex.[12] The "neutral gender" has been arbitrarily defined as comprising all former males and females who have reached an approximate age of sixty years. It must be remembered that this is, of course, a very loose definition in terms of age onset. There are obviously many women who are confirmed members of the "third sex" before they reach their fiftieth birthday. In contradistinction, there are many men who obviously have not qualified for third sex membership by the time they reach their seventieth birthday. It should be remembered that there is roughly a fifteen-year lag in involution of gonadal sex steroid productivity between males and females of corresponding ages. Thus, since we expect the female climacteric to range within the forty to fifty-five year age group, the male climacteric should run approximately from ages fifty-five to seventy.

If we accept the existence of a "neutral gender" from a medical point of view, we are then faced with the problem of recognizing the clinical existence and providing adequate support for this age group during our ever-increasing life span. Primarily, our attention should be directed toward the physiological evidence of sex steroid starvation. Of prime import in this direction is the major concern of the aging process: protein wastage.

The anabolic effects of the sex steroids have been well defined clinically for several years.[13-16] Of recent vintage, however, is the supportive laboratory work of the protein balance studies associated with the long range sex steroid replacement efforts.[17,18]

An excellent example of a specific protein imbalance peculiarly associated with the aging process is that provided by the clinical entity of osteoporosis. In this situation, the bony matrix (a protein material) is dissipated from its normal situation in the marrow cavity, once the individual's sex steroid levels are inadequate to maintain positive nitrogen balance. In the female, the onset of this clinically distressing phenomena is, as might be expected, subsequent to the climacteric in the fifty-five to sixty-year age group. If adequate protein balance is obtained through the anabolic effects of steroid replacement techniques, the bony matrix is returned to its normal consistency, and the continued efforts at calcium deposition become clinically effective. In other words, there is no such thing as old bone.[19] If the bony matrix is maintained at a clinically effective level, calcium deposition will continue effectively in the eight-year-age group. Thus, osteoporosis may well be described as a reversible involutionary process.

It must also be realized that the effects of the sex steroids on body economy, range far from the basic concerns of protein balance and calcium metabolism. As an example of the widespread influence of the sex steroids in multiple physiological processes, we may cite the more recent work on the increased instance of atherosclerosis in castrated women as opposed to a controlled population.[9] The possible effects of the steroids on the cardiovascular system has opened a new field of investigative interest to the research cardiologists.

In the field of senile mental aberrations, the effectiveness of the sex steroid support in the early phases of senile mental involution has been well established. Long range evaluation of patients under steroid support in a controlled series was carried out by the Department of Neuropsychiatry of the Washington University School of Medicine.[20,21] It is apparent that such early involutional symptomatology as the loss of memory for recent events, the reduced capacity for definitive thinking, et cetera, may be effectively reversed, if steroid supportive measures are instituted sufficiently early in the senile mental involutionary process.

It is indeed unfortunate that senile mental involution occurs with extreme rapidity. Thus, the endpoint for reclamation may well be passed long before family and friends are sufficiently alarmed to bring the individual patient to adequate medical attention. It is also true that many sex steroid-supported patients will ultimately go through significant mental involutionary states. However, this is a long-delayed process when compared to the rapid mental disintegration of the unsupported clinical control group of patients.

If we are to continue long range steroid replacement, we must also assume the complete responsibility of protecting the individual woman against any untoward results which might be occasioned by the replacement techniques. We must protect her against such minor distress as the stimulation of secondary sex characteristics, uterine growth and ultimate endometrial breakthrough bleeding, prolonged and irritating breast tenderness, hirsutism, lowering of vocal pitch, and clitoral irritability. There are four major techniques by which sex steroid replacement can be maintained without untoward stimulation of secondary sexual characteristics.

TABLE I. ESTROGEN INFLUENCE OPPOSITION METHODS

1. Cyclic estrogen exhibition and withdrawal at regular intervals to shed the endometrium.
2. Constant estrogen influence with cyclic progesterone exhibition and withdrawal to shed the endometrium.
3. Constant estrogen influence with cyclic testosterone exhibition and withdrawal to shed the endometrium.
4. Constant estrogen influence and testosterone opposition to such a degree that hyperplasia is avoided and shedding of the endometrium unnecessary.

One of the primary concerns of the medical public is the question of the possible carcinogenic effect of the sex steroids. This professional anathema to steroid replacement has been predicated upon the basic belief that endometrial hyperplasia is, in many instances, a precurser to endometrial carcinoma. It is obvious that the procedure of choice in any situation of steroid replacement is to avoid the possibility of developing endometrial hyperplasia. The replacement technique, using combinations of testosterone and estrogen at approximately a 20 to 1 milligram for milligram-absorbed dosage,[22] accomplishes this purpose most effectively.

It is impossible for the uterine endometrium to develop hyperplasia under the 20 to 1 combined steroid influence. Other than a possible vaginal spotting from the fourth to the sixth week under maintained steroid influence, the endometrium will not be the cause of bleeding distress. As a matter of fact, after six months of therapy, the uterine endometrium is completely senile tissue, incapable of breakthrough bleeding. Also under the influence of this combined steroid dosage, there well may be a passing breast tenderness, lasting from three to six weeks under therapy. The patient can be completely assured that, after roughly six weeks of therapy, the breast tenderness will disappear, and will not again recur regardless of how long steroid therapy is maintained. Clitoral hypertrophy or irritability has not been a factor in the 20 to 1 ratio influence on women.

Roughly, 20 per cent of the patients under the combined steroid dosage will have either hirsutism and/or lowering of the vocal pitch. This will be particularly true for those of brunette coloring. If it is apparent that hirsutism is progressing, a steroid combination of roughly 15 to 1 absorbed milligram ratio (testosterone over estrogen) should be the therapy of choice, rather than the previously described 20 to 1 ratio. The main reason for the progressive hirsutism in this particular situation is, in all probability, the corticoid secretory influence of an active adrenal gland during this involutionary phase of life. Lowering of the vocal pitch has not been treated effectively. It simply must be accepted as a minor distress of present replacement therapy methods, when it occurs.

It is important to emphasize that we as physicians, must concede the existence of the "neutral gender," a physiologically established fact. Once the "neutral gender's" existence has been accepted, the responsibility for physical and mental support of the aging population will weigh heavily on our hands. The future of steroid replacement certainly is in the direction of those steroid-like substances, which have little or no secondary sex stimulative characteristics, but do retain protein protection, calcium salvage, atherosclerosis retardation, and protection from senile mental involution afforded by the present steroid combinations as part of their normal physiological function. As physicians we can no longer ignore the responsibility of effectively supporting our aging population.

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Thermal Burns of the Eyelids and Chemical Burns of the Eyes

Emergency Treatment

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BURNS OF THE upper part of the face are of greatest importance in their interference with the visual function. In addition, however, burns of this area cause more cosmetic defect than burns of similar extent elsewhere on the body. To these primary effects we must add the important resultant influence the visual and cosmetic defects have on the emotional and economic status of the patient, his family and his community.

Thermal Burns

Thermal burns of the upper face usually involve the eyelids without burns of the eyeball—only about 12 per cent of patients with burns on the skin of the eyelids have burns of the eyeball.[1] This, of course, is due to the natural protection afforded the eyeballs by the lids in their "winking reflex" closure when potentially injurious material approaches the eyes. The eyeball itself is burned, however, when flames or scalding material spread with explosive force (too fast for reflex closure to operate), or when the heat is so intense or prolonged that parts or all of the lids are destroyed.

When burns of the face are extensive and endanger life, the important systemic treatment is usually the responsibility of the general surgeon and includes the measures to prevent and combat shock (fluid replacement, control of pain and anxiety, maintenance of rest and body temperature, et cetera). Systemic antibiotics and antitetanus treatment are also important.

Initial local treatment to the burned area is directed at the prevention of infection and the restoration of an adequate covering as rapidly as possible before scarring develops and deformity results.[2]

First aid measures should consist of a dry sterile dressing and systemic control of pain. When hospital care is obtained, the wound may be gently cleaned if extremely dirty (vigorous cleaning and debridement are contraindicated), fluid blebs are left intact as additional barriers against infection, and antibiotic ointments may be applied if gross contamination is certain. An initial layer of meshed gauge, impregnated with petrolatum or boric acid ointment, is placed over the wound and then a thick tight fluff gauze dressing is applied to provide gentle compression and an absorbent material for the fluid wound exudate. Such a dressing in extensive facial burns may necessitate a complete envelopment of the head except for the nose and mouth. If the eyeball has not been damaged, an ointment may be applied to the eye and the eye left covered until the general dressing is changed.

The use of oils, oil-soaked cloths, or greasy materials as a first aid measure should be discouraged for burns about the eyes as they are for burns elsewhere. These are almost certain sources for contamination and make the later definitive wound treatment more difficult.

The surface coagulants (picric acid, tannic acid, gentian violet, and triple dye) are not used extensively for burns about the eyes. As with general body burns, it is recognized that these agents interfere with new skin growth, actually increase the scarring, and infection is frequent under the "protective covering." The use of the more recent plastic spray coverings is also being discouraged because of interference with new cell growth.

Burns are classified according to their depth as first degree (simple erythema), second degree (bleb formation—partial destruction of skin with survival of some of the epithelial cells), and third degree (destruction of the full thickness of the skin).

Presented at the regional meeting of the American College of Surgeons, St. Paul, Minnesota, April, 1957.

In the partial-thickness burns where healing occurs from the surviving epithelial cells, antiseptics should not be used because they, too, interfere with cell growth. A suitable initial dressing is necessary for protection from contamination and further injury. The deeper second-degree burns heal with some scarring; the first degree and superficial second-degree burns heal without scarring. In whole-thickness (third-degree) burns and some deep second-degree burns, the convalescence is speeded up and the contractures and late deformities are minimized by skin grafting as early as possible. The most critical area for this early grafting is the skin of the eyelids.

Most of the thermal burns which involve the eyeball show only mild erythema and require relatively simple treatment such as a mild antiseptic ointment (e.g., White's mercury bichloride ointment) and possibly a cycloplegic (e.g., atropine) if the inflammation is marked. Immobilizing the lids with a pressure dressing usually makes the patient more comfortable and also, because of the higher globe surface temperature under the closed lids, promotes faster healing.

Small areas of more severe burns of the palpebral and bulbar conjunctiva and cornea are adequately treated with lubricating ointments and repeated passage of a blunt probe along the fornix to prevent the formation of symblepharon.

Extensive deep burns of the conjunctiva are best treated by immediate mucous membrane graft. This graft may be conjunctiva from the other eye or buccal mucous membrane (lower lip or either side of the mouth). Buccal mucous membrane is not as satisfactory in an exposed area of the globe because it remains permanently somewhat thickened and red.

Deep burns involving the cornea will inevitably result in some degree of scarring and loss of vision when the scarring is central.

The use of cortisone cuts down irritability of the eye and reduces the density and vascularization of the scar.

Chemical Burns

A chemical burn of the eye is defined as an injury resulting from local contact with a chemical (solid, liquid, dust, mist or vapor) to such a degree that there is alteration in the structure of the cornea and conjunctiva. If this alteration is not visualized grossly, it may be demonstrated by a positive stain using 2 per cent solution of fluorescein.

Although this type of eye injury occurs with almost all activities of life, industry is responsible for the great majority of them. The industries where this type of injury is particularly prevalent have adopted measures for prevention and have established facilities for the important quick first aid treatment.

Not long ago the immediate treatment advocated for a chemical eye injury was to inactivate the injurious chemical with a specific prepared material (for example, acid for alkali, and alkali for acid). In order for this ideal in treatment to be effective, the inactivating material itself must not damage the tissue, it must penetrate the tissue as rapidly as the injuring chemical, and it must be ready for immediate use before irreversible damage is done. Because these criteria rarely can be satisfied, any attempt to treat a particular chemical eye injury with a specific neutralizing material is now considered detrimental.[3]

There are two exceptions to this rule. One, the treatment of lewisite burns with the specific agent, BAL (British Anti-Lewisite or dimercaptopropanol). This is the only effective treatment against this arsenical war agent and is also effective for several types of arsenical poisoning encountered in civilian practice. Another exception is the commonly used cocaine hydrochloride for neutralizing the iodine used as a cauterizing agent in the treatment of dendritic (herpes simplex) infection of the corneal epithelium.

The immediate treatment now generally accepted for all types of chemical eye injuries is the quick irrigation of the eye with *water at the nearest source of supply.* The industries where chemical eye injuries are common familiarize the new employee with the location of these water sources and with the importance of immediate and thorough irrigation. Some industries also have special eye irrigation water fountains, operated by the foot, placed strategically in the plants for immediate and very effective irrigation. This "on the job" water irrigation is advised for a period of about five minutes and then the patient is conducted to the physician where, after instillation of a local anesthetic, irrigation with water or normal saline is continued for about half an hour. Also, during this period the eye is carefully inspected, under loupe or slit-lamp magnification, and any insoluble particles on the ocular surfaces are removed using applicators or forceps.

The initial phase of irrigation removes any unreacted portion of the injurious agent still present on the corneal and conjunctival surfaces. Continued irrigation is necessary to remove the chemicals which have already combined with the tissues and begun their denaturing process.

In addition to immediate and prolonged irrigation, mechanical removal of injured or possibly contaminated tissue (vigorous wiping of the cornea and conjunctiva with cotton applicators) has been advocated.[4] This, however, has not been accepted generally as more effective than adequate washing.[3]

After the measures for removing the chemical have been exhausted, attention is directed toward providing conditions favorable to healing and toward prevention of complications such as infection and formation of conjunctival adhesions. An antibiotic ointment is used both to prevent infection and also to provide a lubricant to prevent adhesions. Continued use of local anesthetics is avoided because of their detrimental effect on epithelial regeneration. A cycloplegic is indicated for any associated iritis (atropine for the more severe cases). Firm eye dressings are usually advisable for the comfort of the patient, in order to promote healing from the increased corneal surface temperature, and to avoid mechanical disturbance of the regenerating epithelium. Also, the use of cortisone on chemical burns, clinically and experimentally, has been shown to reduce the inflammation, scarring and corneal vascularization.[5] Cortisone should not be used, however, in the presence of uncontrolled infection.

After the initial examination and treatment, it is important to know the identity or chemical nature of the substance causing the injury. When we know this and also have a familiarity with the type of ocular injury to be expected from the class of chemicals to which the particular substance belongs, we are aided in predicting the probable course, prognosis, and extent of treatment necessary.

The injurious action of both acids and alkalies is fundamentally a result of the shifting of the hydrogen ion concentration of the tissue out of the range in which the tissue components are stable.[3] Other ions play an additional role in some instances but in general the injury from acids and alkalies depends on the concentration of hydroxyl and hydrogen ions and the duration of their action.

With acids of considerable strength the action is one of coagulation of all protein with which contact is made thus forming insoluble acid proteinates. This reaction is irreversible and so the destruction of the affected tissue is permanent. The destruction is also instantaneous. Penetration of the acid is limited by the barrier made by the dense layer of precipitated protein. A whole layer such as the cornea may be lost only when the injuring acid is great in concentration and amount.

Alkalies produce some of the most severe chemical eye injuries.[2] The increase in hydroxyl ion concentration beyond the limits of tissue protein stability results in the formation of gel-like alkaline proteinates. In addition to this, however, the alkalies combine with the fats to form soaps and in this way they destroy the structure of the cell membranes and thus penetrate rapidly into the tissues. This speed of penetration is responsible for the capacity of alkalies to cause great intraocular damage. For example, traces of ammonia were detected in the anterior chamber as early as five seconds after liquid ammonia was placed in the rabbit's eye.[2] Alkalies also have a hygroscopic action which removes water from the cells and adds to the total necrotic process.

There are also some chemicals which produce changes in the tissue proteins but do not significantly alter the hydrogen ion concentration. The altered protein is unable to perform its function and therefore inflammatory and degenerative reactions result. The injurious effect on the eye from such a chemical may be just as severe as from alkalies and acids. Examples of these are the war gases, lewisite and mustard gas.

After the initial immediate irrigation and cleaning necessary for all chemical eye injuries, a study should be made of the chemical properties of the injuring substance.

This is particularly important now that there is such a great variety of products being used in industry and the home . . . with new chemicals being added daily. When these properties are known we are aided in planning future treatment and predicting the prognosis.

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BCG Vaccination in a Tuberculosis Prevention Program

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THE EXISTENCE of immunity in tuberculosis has been challenged frequently, although it has been shown unequivocally in animals and man that tuberculous infection which is well controlled does produce appreciable immunity against a tuberculous reinfection. It has been stated recently that most cases of tuberculosis are derived from those individuals who are positive reactors to tuberculin. On the face of it, this is a true statement since one cannot have disease without reacting to tuberculin, but the implications are misleading. One cannot make this general statement without qualifications.

For example, a positive tuberculin reaction in an infant, child or even a very young adult may mean a recent infection. This is especially true when such individuals react to a small dose of tuberculin. In such groups, the course of events may be either one of healing or of active disease. Naturally, under these conditions the majority of the cases of tuberculosis will arise from this group. However, if a tuberculous infection has gone on to healing as one is more likely to find in adults, but can also be found in children, such an individual is more resistant to a reinfection than a person who has never had an experience with the tubercle bacillus and later becomes exposed to virulent infection.

Bates and Davey,[1] University of Michigan, found that in a ten-year observation period of medical and nursing students there were twenty cases of tuberculosis ranging from minimal to moderately advanced with cavity formation. All of these occurred in students who were negative reactors to tuberculin on admission to school (first and second strength PPD). There were no cases of tuberculosis in the students with positive tuberculin reactions at the start of training. All the twenty cases occurred in students who failed to react to even 0.005 mg. of PPD.

The well-documented British study in adolescents[2] revealed an incidence of 0.74 per 1000 per year among those originally positive to 1:100 O.T. (100 TU) only, as compared to 1.94 per 1000 per year in a concurrent negative reactor group. Among those who were initially positive (15+mm) to 1:3000 O.T. (3 TU) the rate was 2.93 per 1000 per year. Thus, there was up to four times as much tuberculosis in those who reacted to the small dose of tuberculin or did not react at all, as compared to those who reacted to the large dose of tuberculin (100 TU) only. Recently, the U. S. Public Health Service found a similar correlation in their study in student nurses. They found approximately one-half as much tuberculosis in those who reacted to the larger dose of tuberculin only as compared to those who reacted to the small dose or not at all. As will be discussed below, those reacting to the large dose of tuberculin had practically the same degree of immunity as those vaccinated with Bacillus Calmette-Guérin.

Another striking example of acquired resistance is the experience of the Papworth Village (England).[3] Here, parents with tuberculosis are allowed to live a normal communal life with their children under more or less ideal conditions (food, housing, security, and so forth). Of the 108 children born in the village since 1921, Dr. R. R. Trail, present director, writes (1955): "We do not know of any proved case of clinical pulmonary tuberculosis to date, and yet we know that all these children were tuberculin-positive in childhood, the majority by the age of two and one-half. Seventy of the boys were captured in Singapore and worked on the Death Railway of Siam. None of them developed tuberculosis.[4]

In summarizing, then, a healed primary infection in human beings in the lung or elsewhere confers an increased resistance against virulent reinfection. This is attested by a decreased incidence of disease, a greater localization, and a better prognosis. Paradoxically, however, even

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in healed infection, the possibility that viable organisms may produce active disease at a later date is present, since in many instances they may remain viable in the body. It is for this reason that artificial immunization with an organism such as BCG, which can be controlled, would be highly desirable when its potentialities as a vaccine, though not as great, approach those of the virulent organisms.

In regard to BCG vaccine, recent advances in freeze-drying have made it possible to standardize this vaccine (viability, potency, et cetera), as well as determine its safety before distribution. In fact, at the present time the vaccine as produced bv Research Foundation and the University of Illinois is not distributed until all the standardization and safety tests have been completed. This usually takes a period of six months.[5] The recent report by Dubos and Pierce has indicated that the freeze-dried vaccine distributed by Research Foundation and the University of Illinois multiples readily in his mice, that it is a good cord former, that the freeze-dried form is as effective as the fresh vaccine, and that it compares favorably with other vaccines in this country or abroad.[6] Dr. Willis and Dr. Vandiviere of Chapel Hill have shown that this same vaccine is as potent or more so in animals and man than any other vaccine, including BCG, they have tested.[7] This has also been verified by Donald Smith of Madison,[8] Wisconsin and Guy Youmans of Northwestern University.

In summary, BCG in the dried form, as produced by Research Foundation and the University of Illinois, is a highly effective vaccine which is well standardized before distribution.

Is BCG effective in the prevention of tuberculosis in humans?

The recent clinical studies in regard to the efficacy of BCG vaccine is most impressive. In May, 1957, Dr. Aronson presented his Indian studies followed for a period of twenty years at a meeting of the American Trudeau Society. There were three times as many cases of minimal pulmonary tuberculosis, ten times as many cases of moderately advanced tuberculosis and three and one-half times as many cases of far advanced tuberculosis in the controls as in the vaccinated. There were five times as many tuberculosis deaths in the controls as compared to the vaccinated. All but fifteen of over 3,000 individuals under study were followed.

One of the best controlled studies was initiated in 1950 by the Tuberculosis Vaccine Clinical Trials Committee of the Medical Research Council of Great Britain and included nearly 56,000 male and female students in secondary schools between fourteen and one-half and fifteen years of age, since the incidence of tuberculosis in Great Britain rises markedly at the age of fifteen. The experiment was carried out in the heavily populated areas of North London, Birmingham and Manchester. Those children who had known contact with a case of tuberculosis within two years were excluded from the trial.[2]

All eligible children were given tuberculin tests and chest x-rays were taken. The first tuberculin test consisted of 0.1 ml. of a 1:3000 dilution of Old Tuberculin (3 TU). If negative to this strength, they were retested with 0.1 ml. of 1:100 dilution of Old Tuberculin (100 TU). Those children found to be negative to the largest dose of tuberculin were divided into two groups according to the final digit of the serial number appearing on their record cards. The children of one group were vaccinated intradermally with 0.1 ml. (0.075 mg.) of a freshly prepared liquid vaccine which was obtained from the state Serum Institute in Copenhagen. Nearly 94 per cent of the participants were followed for the first thirty months and a somewhat lesser number were followed over a period of four years. (The children were contacted three times and final tuberculin tests and chest x-rays were given.) The films were reviewed by an independent assessor who was kept unaware of the results of the tuberculin test and whether or not vaccination had been performed.

During the first two and one-half years, there were thirteen cases of tuberculosis among the BCG-vaccinated group which gave an annual incidence of 0.37 per 1,000 and there were sixty-four cases in the tuberculin negative non-vaccinated group, an incidence of 1.94 cases per 1,000 children. During the period between two and one-half and four years after the start of the study, thirty-eight additional cases occurred in the unvaccinated group, and five in the BCG-vaccinated group. This indicated that BCG *vaccine* retained its effectiveness for as long as four years.

TABLE I. MEDICAL RESEARCH COUNCIL (GREAT BRITAIN) STUDY

Cases of tuberculosis starting within two and a half years of entry to the trial*

Section		Skin Test and Vaccination Group	Estimated Number of Participants	Definite Cases of Tuberculosis		Possible Cases of Tuberculosis Starting Within 30 Months
				Number Starting Within 30 Months	Annual Incidence Per 1,000 Participants	
A	Children admitted concurrently with those given BCG vaccine	Negative unvaccinated	13,200	64	1.94	2
		Negative, BCG, vaccinated	14,100	13	0.37	2
		Positive to 3 T.U.	15,800	69	1.75	3
		Positive only to 100 T.U.	6,500	12	0.74	1
B	Children admitted concurrently with those given vole bacillus vaccine	Negative unvaccinated	6,400	33	2.06	2
		Negative, BCG, vaccinated	6,400	5	0.31	1
		Negative, vole bacillus vaccinated	6,400	7	0.44	1
		Positive to 3 T.U.	8,600	37	1.72	2
		Positive only to 100 T.U.	3,500	6	0.69	0
C	All participants included in the above comparison†		56,000	165	—	9

*For the definition of the starting point of the illness, see text.

†That is, all participants in Section A plus the vole-bacillus-vaccinated group in Section B.

There can be no question in this study but that the reduction in the incidence of tuberculosis was due to BCG. The random allocation of children to the two groups, the freedom from bias in the assessment of the occurrence of cases and the severity of cases make any other conclusion impossible. One statement on the efficacy of BCG vaccination in this study reads as follows: "According to the present results, if none of the tuberculin-negative entries had been vaccinated, 165 cases of tuberculosis would have been expected among them within thirty months of entry. If all of them had received BCG vaccine, thirty cases would have been expected. The difference of 135 cases represents a reduction of 82 per cent in the incidence of tuberculosis in the tuberculin negative group (Table I).

In our own studies in Chicago (which included all age groups in both white and colored individuals) we found that in newborns with no contact with tuberculosis but coming from areas where the incidence of tuberculosis is high, that there was a 78 per cent reduction in morbidity and 79 per cent reduction in the mortality in the vaccinated as compared to the control group. This group was followed for eighteen years. In babies born to tuberculous households (followed for 15 years) in which all the children were isolated in foster homes for varying periods of time, there was a 68 per cent reduction in the morbidity and an 80 per cent reduction in mortality in the vaccinated as compared to the control group (Table II).

In children in a federal housing project followed for thirteen years, where a complete tuberculosis control program was in effect, there were no cases of tuberculosis in the vaccinated and nine cases in the nonvaccinated, with two deaths. This latter group included those who did not react to tuberculin, three cases; those who reacted to tuberculin, three cases; and those in whom the tuberculin status was unknown, three cases. In medical students at the University of Illinois followed over a period of sixteen years there were no cases of tuberculosis in the vaccinated as compared to ten cases in the non-vaccinated (two in the negative reactors, four in the positive reactors and four in whom the tuberculin status was unknown). In nursing students at the Cook County Hospital over a fifteen-year period there was a 58 per cent reduction in the morbidity of the vaccinated as compared to the controls. However, the vaccinated were allowed to work in a tuberculosis hospital for two months whereas the negative-reactor controls were not allowed to do so.

A comparison of the morbidity and mortality rates of all Chicago projects mentioned, reveals that the highest morbidity rate is found in those groups in which the exposure is the greatest, namely, nursing students and newborn infants in contact with tuberculosis. This morbidity is high irrespective of the fact that in both of these groups the housing, nutrition, and the social status were of a relatively high order. Vaccination reduced the morbidity 58 and 78 per cent respectively, compared with the controls under similar conditions, except as indicated.

Improvement in housing favorably affects the morbidity rate in children not vaccinated and

TABLE II. RESULTS OF STRICTLY CONTROLLED STUDIES OF BCG VACCINATION

All Age Groups Carried Out Over Past Eighteen Years

	Vaccinated					Non-Vaccinated					Per Cent Reduction in Vaccinated Group[1]	
	No. in Study	Tbc. Cases	Morb. Rate[2]	Tbc. Deaths	Mort. Rate[2]	No. in Study	Tbc. Cases	Morb. Rate[2]	Tbc. Deaths	Mort. Rate[2]	Morbidity	Mortality
Infants												
Non-contact	5426	18[3]	3.3	2[3]	0.4	4128[4]	63	15.3	8	1.9	78	79
Contact	311	4	12.8	1	3.2	250[4]	10	40.0	4	16.0	68	80
Children												
Orphanages	2370	0		0		1348[5]	12	8.9	0		100	
Housing project (Ida B. Wells)	947	0		0		944[4]	3	3.2	0		100	
						875[5]	3	3.4	1	1.1	100	100
						584[6]	3	5.1	1	1.7	100	100
Housing project (Altgeld)	2574	0		0		1900[5]	0		0			
						612[6]	0		0			
Adults												
Student nurses	269	2	7.4	0		281[4]	5	17.8	0		58	
						476[5]	3	6.3	0		—17	
Medical students	472	0		0		313[4]	3	9.6	0		100	
						1079[5]	4	3.7	0		100	
						279[6]	5	17.9	0		100	

[1]—Rate for control group taken as 100 percent.
[2]—Rate per 1,000.
[3]—One infant had contact since birth with open tbc. The other, a "no take" after vaccination.
[4]—Negative tuberculin reactors.
[5]—Positive tuberculin reactors.
[6]—Tuberculin reaction status incomplete.

non-reactors to tuberculin (3.2 per thousand in contrast to 15.3 per thousand in poor housing areas). Complementing good housing with BCG vaccination lowered the morbidity rate to zero. These figures stress the importance of housing in a tuberculosis control program. When a complete tuberculosis control program is in effect, including removal of persons with active tuberculosis, providing adequate housing and nutrition, plus periodic examinations, the incidence of tuberculosis in those vaccinated with BCG was zero.

In summary, BCG has been shown unequivocally to increase the resistance against tuberculosis in numerous human trials. No vaccine has had better valid statistical evidence to prove its efficacy in human beings. BCG should be considered only as part of an overall tuberculosis control program.

Dr. Geoffrey Edsall,[9] editor of the *Journal of Immunology* and Dr. Guy Youmans, head of the Department of Bacteriology, Northwestern University,[10] recently stated that there is more statistically acceptable evidence that BCG is an effective vaccine against tuberculosis than there is for diphtheria and tetanus toxoids and even smallpox vaccine.

Will the usefulness of the Tuberculin Test be lost as a diagnostic measure?

In the diagnosis of active tuberculosis the value of the tuberculin test is not paramount, since various laboratory tests including determination of sedimentation rates and making of x-rays are used to make the final diagnosis. However, the possibility that the usefulness of the tuberculin test might be lost to the epidemiologist must be considered. This is particularly true in areas where the rate of tuberculous infection is low. Under these conditions, the detection of a small proportion of the infected individuals by means of the tuberculin test becomes worthwhile. Where the incidence of tuberculous infection is high, however, as in certain sections of many of our large cities, the determination by means of the tuberculin test that the majority of the population is infected is of little value. The appropriate control measures can be applied just as easily to the whole population as to the majority found to be tuberculin-positive. The question should be considered seriously whether an 80 per cent reduction in the incidence of tuberculous disease in vaccinated individuals might not more than compensate for the lack of the usefulness of the tuberculin test for the determination of the incidence of infection in those localized areas of high prevalence.

With chemoprophylaxis, is vaccination necessary?

To answer this question, one must distinguish between the management of those individuals with negative tuberculin reactions and the management of individuals who are recent converters.

Persons with Negative Reactions.—In the management of those with negative tuberculin reactions, there is a choice of using BCG or chemoprophylaxis. BCG is preferable because: (1) it raises the patient's immunity to tuberculosis, (2) its effect lasts for several years, (3) it has had a large scale trial, and (4) after vaccination it is effective without further co-operation on the part of the patient. Chemoprophylaxis on the other hand: (1) does not raise immunity by itself, (2) suppresses tubercle bacilli only during the period the drug is taken, (3) is experimental, and (4) requires continued patient co-operation over a period of many months.

"Recent Converters."—Isoniazid has been suggested for "recent converters," especially in infants under two years of age and for nurses. It is recommended that treatment be given daily and continued for one year. This method should not be considered as a substitute for use of BCG but as an adjunct to it; that is, in converters who have not been vaccinated or in vaccinated persons who have a marked augumentation of their tuberculin reaction, and particularly in all persons with x-ray evidence of the disease. In March, 1956, Waring, who was among the first to recommend the treatment of recent converters with isoniazid stated:

> "I think negative reactors to the tuberculin test among highly exposed persons should have BCG vaccination. This would include nurses, medical students and children in tuberculous families. On the other hand, if you do not approve of this, one must surely seriously consider chemotherapy of recent converters with or without x-ray pulmonary signs."

It is understandable that one may expect even greater difficulties in the administration of a chemoprophylaxis program aimed against tuberculosis among civilians than was experienced under military conditions with the chemoprophylaxis of malaria and gonorrhea.

The possibility of the development of resistance to isoniazid by the infecting organism must also be considered. Furthermore, it is known that before tuberculin sensitivity develops, the infecting organism probably has already multiplied, disseminated and produced pathology. Dr. Edith M. Lincoln of New York University, Bellevue Medical Center, states: "In a study over a period of about fifteen years, just before chemotherapy, we found that over 85 per cent of children under two years of age with positive tuberculin test showed either parenchymal or node involvement or both." (It should be emphasized that this was a group of consecutive cases all found on wards of a pediatric service.) Since isoniazid does not kill the virulent tubercle bacilli in the animal host, these organisms will remain as a constant source of danger. Every effort should be made to minimize the multiplication of virulent organisms within the body.

Conclusions

BCG is an effective vaccine for the prevention of tuberculosis. It should never be considered as a substitute for the time-honored and time-tested principles of tuberculosis control but as an adjunct to these methods. BCG vaccination is recommended for infants and children in areas of high incidence and for those individuals who will be unavoidably exposed to tuberculosis (medical and nursing students, individuals in tuberculosis households, penal and mental institutions, et cetera).

These recommendations have been made by the American Trudeau Society, which is the medical branch of the National Tuberculosis Association, and the U. S. Public Health Service, and have been recognized by the Council on Drugs of the American Medical Association.

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Problems of Selection in Studies Based upon Hospital and Autopsy Data

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THE STUDY of association has proved invaluable in medicine. The philosophy of diagnostic medicine is based upon the utilization of known associations between certain signs, symptoms or syndromes and particular diseases. Much of our understanding of the mechanism of disease has come from the observations of characteristics of persons who get the disease. Persons with certain characteristics exhibit an apparently higher incidence-rate or more severe manifestations of the disease than others.

Investigations which attempt to discover such associations are almost always based upon the observation of a series of patients who have presented themselves at a particular institution or group of institutions. These institutions are usually the larger hospitals or clinics in a given area. They are the ones with the staff, money and research goals necessary to keep adequate records and develop a large enough series of cases.

The cases represented by an autopsy series are certainly not typical of all deaths. The majority of deaths do not result in autopsy. What forces select a particular death for any given series? In any series of hospital patients the individuals have proved themselves different from the general patient by arriving at that particular hospital for treatment rather than at another hospital. What brought them into this investigator's series?

The effect that selective forces may have upon hospital data studies and autopsy data studies has been discussed before by Berkson[1] and Gilliam.[2] The purpose of this paper is to reemphasize the importance of these selection forces and to demonstrate by way of two hypothetical examples, the biases they may create. A more general discussion follows the example.

Examples

Example 1.—An association between rheumatoid arthritis and rheumatic heart disease has been observed by several investigators. Most of the articles concerning this apparent association have been supported by a set of data based upon a series of necropsies. Each investigation points out essentially the same thing. Among persons with a diagnosis of rheumatoid arthritis, evidence of rheumatic heart disease occurred much more frequently than among control groups. The following discussion will be concerned with interpretation of the observed data only. Whether the theory can be justified upon a purely physiological-pathological basis is a subject for another paper.

The paper by Sokoloff[3] was chosen for illustrative purposes only. Many of the others listed in his bibliography would do as well.

TABLE I. INCIDENCE OF EVIDENCE OF RHEUMATIC HEART DISEASE IN AUTOPSY GROUPS

	Evidence Rheumatic Heart Disease	No Evidence Rheumatic Heart Disease	Total	Percent with Evidence
Rheumatoid arthritis patients necropsies	10	91	101	9.9
Consecutive necropsies (19 or more years of age)	66	1088	1154	5.72
	76	1179	1255	

The author defines as precisely as possible what he considers as evidence of rheumatic heart disease at autopsy. He gives the source of data and summarizes the results in a table like Table I.

After discussing some of the limitations of the material, the author states, "If recognition is made of all of these limitations, the results of the present investigation suggest that in the individuals with rheumatoid arthritis, there may be a somewhat higher incidence of heart disease indistinguishable from rheumatic heart disease than chance would dictate." The statement is essentially correct. Unfortunately, it is a statement about the observations upon a group of selected individuals observed in the autopsy series of these particular institutions. There is a very real possibility that the reason for the observed high incidence of heart disease among the rheumatoid arthritic patients has nothing at all to do with a relationship between the two diseases in the unselected population but is entirely the result of the selection which brings the patient to autopsy.

Bayles[4] writing on this same subject gives a clue to the possible explanation of these observations. ". . . why they (rheumatoid arthritics with rheumatic heart dis-

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TABLE II.
SUMMARY OF HYPOTHETICAL AUTOPSY RATES

Diseases Diagnosed at Death	Autopsy Rate
Rheumatic heart disease	40 percent
Rheumatoid arthritis	10 percent
Rheumatic heart disease and rheumatoid arthritis	50 percent
Rheumatic heart disease and all others (average)	60 percent
Rheumatoid arthritis and all others (average)	30 percent
All others (average)	20 percent

TABLE III. INCIDENCE OF EVIDENCE OF RHEUMATIC HEART DISEASE IN POPULATION

	Evidence Rheumatic Heart Disease	No Evidence Rheumatic Heart Disease	Total	Percent with Evidence
Rheumatoid arthritis diagnosis patients	20	980	1000	2 percent
Other patients	20	980	1000	2 percent

TABLE IV. EXPECTED NUMBER OF EACH TYPE OF PATIENT AT AUTOPSY

	Evidence Rheumatic Heart Disease	No Evidence Rheumatic Heart Disease	Total	Percent with Evidence
Rheumatoid arthritis diagnosis patients	(.5)(20)=10	(.1)(980)=98	108	9.9 percent
Other patients	(.6)(20)=12	(.2)(980)=196	208	5.8 percent

ease) should come to autopsy more than an unselected group (of rheumatoid arthritics) is not clear, unless the type of fatal illness led to hospitalization and more intensive study. It is well known that sufferers of rheumatoid arthritis do not die of this disease and many die of other causes in nursing homes and in their own homes where facilities for autopsy examinations are not readily accessible." If one admits the possibility that a patient who has a dramatic disease or one wherein the death might be sudden and unusual has a greater chance of arriving at autopsy than others do, then a simple possible answer to Bayles' dilemma presents itself.

Suppose that 2 per cent of all persons would have evidence of rheumatic heart disease observable at autopsy regardless of whether they are persons with rheumatoid arthritis or just drawn from the population in general. Suppose, further, that the general autopsy rate for the entire population under consideration is about 20 per cent. The general autopsy rate is contributed to by heart disease deaths, cancer deaths and accidental deaths, all of which will have high autopsy rates, as well as deaths among persons with diseases less apt to be investigated by subsequent autopsy. It would seem reasonable, then, that the autopsy rate for persons with rheumatoid arthritis alone would be considerably lower than the autopsy rate for the population in general, say about 10 per cent. It would also seem reasonable that those persons with rheumatic heart disease damage would have a higher autopsy rate than those from the average population, say 40 per cent.

Finally, a patient may have two or more diseases present and diagnosed at the time of death. It would seem reasonable that this would increase the likelihood of autopsy above that of a patient having but one of the diseases. Such a multi-disease patient would be more interesting from the scientific point of view and would be more apt to be studied intensively. Table II presents a summary of the assumptions concerning autopsy rates.

Now let us follow 1,000 recent deaths among rheumatoid arthritis patients and 1,000 recent deaths among patients with other diseases. By virtue of our first supposition concerning the incidence of evidence of rheumatic heart disease, the expected incidence for both groups is 2 per cent. A summary of population incidence is given in Table III.

The true picture in the population is clear. What happens to the autopsy data after these selection factors (different autopsy rates) have operated? Imposing the autopsy rates summarized in Table II on the various types of patients listed in Table III gives the expected number of the original 2,000 patients which will be classified in each way at *autopsy*. These are summarized in Table IV.

Obviously the choice of rates utilized for this example were deliberately chosen so that the tabled results would correspond with the data reported by Sokoloff. An attempt was made to make the choices reasonable (2 per cent incidence of evidence of rheumatic heart disease, 20 per cent average autopsy rate). The actual magnitude of the rates chosen may be quite far from the truth. The basic argument will not be altered by such errors, however. The only purpose of the hypothetical demonstration was to show that such selection *could* take place. Furthermore, if one thinks that the assumption of differential autopsy rates is a reasonable one, he must expect such selection forces to create artifacts in such data. The effects of these forces superimposed upon the data, will mask whatever results might have been available and will always lead a critical observer to be suspicious of any unique interpretation placed upon the data.

The fact that several authors have been able to get essentially the same type of results does not constitute any supporting evidence about the relationship between the two diseases. It may simply constitute evidence that the same selection factors were operating in all of their separate experiences. Obviously an experiment is necessary which is designed to determine whether there is an association between the two diseases without being influenced by these selection factors. One valid method for attacking this problem is immediately apparent. A random selection of persons with rheumatoid arthritis should be made and followed until death, with a subsequent autopsy on every one. Then a random selection on some control group should be made and these, as well, followed to death with an autopsy on every one. The comparison of the incidence of evidence of rheumatic heart disease in these two groups would constitute a legitimate study. In such a study the individuals have not been given an opportunity to select themselves into an autopsy series. As difficult as this study may seem it is probably the easiest reliable method for obtaining the answers desired.

Example 2.—Many investigators have reported that the incidence of breast cancer in the family history is higher for patients who have breast cancer than for an arbitrarily chosen control group with a benign disease. Jacobson[5] reports a series of cases where the incidence of breast cancer among mothers of breast cancer patients and benign disease control patients were observed. These data were later given a careful interpretation by Busk.[6] A final interpretation would indicate that the incidence of breast cancer among mothers of breast cancer patients was twice that of the mothers of control patients.

These results are reproduced by several authors. The exact incidence of maternal history of breast cancer is not important for this paper. It is the possibility of selection which is under investigation. It is entirely possible to get results like those observed by permitting the patients to select themselves into the study.

Assume that about 5 per cent of the women in the entire population under consideration have mothers with a previous history of breast cancer. For a given year, start with a hypothetical group of breast cancer patients, say 20,000. According to our previous assumption 19,000 of these breast cancer patients will have mothers with no previous history of breast cancer. One thousand will have mothers with previous history. Let us assume that among the women whose mothers have history of breast cancer, 2 per cent of them will arrive at hospitals concerned. Among the women whose mothers have no previous history of breast cancer 1 per cent will arrive at the hospitals concerned. One per cent of the 19,000 women who have breast cancer but whose mothers had no history of breast cancer is 190. Two per cent of the 1,000 women with breast cancer whose mothers had history of breast cancer is twenty. Under these assumptions we will end up with 210 breast cancer patients at the hospitals concerned, of which twenty have mothers with history of breast cancer. This would lead us to believe that twenty out of 210 (approximately 9.5 per cent) patients hospitalized for breast cancer have mothers with history of breast cancer.

This high rate of family incidence of breast cancer (9.5 per cent) as opposed to the true value of 5 per cent has nothing to do with a hereditary factor. It has nothing to do with the fact that these women have breast cancer. It has to do with selection and entry rate into the hospitals. The entry rate for women with maternal history of breast cancer into these particular hospitals has been assumed to be twice as large as the entry rate for those with no maternal history of breast cancer. Some possible indications for this last assumption are as follows: (a) The awareness of the disease to be found among women with maternal history of breast cancer make for earlier recognition and for their getting to the doctor in time so that treatment is feasible. They are not kept at home as terminal cases without ever arriving at the hospital. (b) The awareness of the disease and its serious implications makes for a desire to go to the leading institutions in the areas and seek the most up-to-date treatment. One may argue that these forces are not sufficient to double the entry rate for one group. One cannot argue that these forces are nonexistent. They are there to a certain extent, however large.

Summary

Two examples have been presented. They represent examples of possible misinterpretation of observed data because of selection bias. The point has been made that these patients must have been patients at the hospitals concerned before they could become contributors to the study.

If the goal of the study is to obtain estimates of incidence in the general population, the possibility of selection must be considered. All that is necessary to invalidate such a study completely is that persons with and without the characteristic being studied should have different entry rates into the study.

If the goal of the study is to describe what is to be seen by a doctor in one of these hospitals (not a description of the population in general), then this type of study may be valuable. It will describe the incidence to be observed after the selection has taken place. Unfortunately, these selection factors will vary from place to place and from time to time. The usefulness of the information would therefore be limited to the source hospital or hospitals.

In general, the solution to this dilemma is to anticipate the possibility of the presence of selection forces. The study must be designed in such a way that the patients are not given this opportunity to select themselves. This may involve the large, more difficult "forward" study. It may involve intensive "follow-up." It may require the cooperation of several hospitals so that an entire area is covered by the survey. Whatever it involves, it can give answers. It is true very often that the quicker, easier methods of study cannot possibly give correct answers.

These selection forces should be expected in all investigations of this kind. A careful investigator will try to anticipate such biases before the study is undertaken and obtain data in such a manner that their influence is eliminated. If this is not possible, the strength of these selection forces should be evaluated so that some estimate of their influence is considered. In any case, the

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Criteria for Pediatric Referral to Psychiatry

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BY WHAT criteria should the busy practitioner be guided in considering pediatric referral to psychiatry?

A few weeks ago a boy of fourteen years was referred for psychiatric attention because of fatigue. This boy had been successively treated for thyroid dysfunction, anemia, possible poliomyelitis, cerebral palsy and damage to the brain—five serious diagnoses and six medication programs. However, the only physical complaint had been that of fatigue; there never had been abnormal results of laboratory procedures. In retrospect, it would appear that the physician who saw the boy at the age of six years should have recognized the symptom of fatigue as reflective of a childhood psychologic withdrawal. Psychiatric treatment was indicated at the time the boy was six years old, before the ravages of poor adjustment to school, unhappy social experiences and parental frustration had added their toll to the emotional war already present in this boy's personality. Yet it may well be that this child's physician had, in fact, recognized the psychiatric need, but had met family opposition in the form of complete refusal to consider a psychiatric diagnosis, insistence on additional "organic" tests and medication programs, or even insults to the physician for suggesting such a degrading thing as "insanity" in the family.

The problems of parental education about psychiatry and the interpretation of defenses used to counter the suggestion of psychiatric referral cannot be the subjects of this paper. A word is in order, however, about a certain technic all too frequently used by physicians. Because of the personal and social bias against psychiatry even the well-intentioned practitioner may find himself asking a patient, "Would you be *willing* to see a psychiatrist?"

This is in startling contrast to other referrals in which the comment invariably goes, "You *should* see a specialist in urology or bone disease."

What is heard by the patient and family when a psychiatrist is mentioned is far more than simply a difference in words; it is the contrast between a physician's offering hesitantly and apologetically the services of a specialist of whom he himself is a bit wary, and one who refers the patient to a colleague for whom respect has been enhanced by understanding and professional interchange. Referrals will not be carried out adequately, no matter how clearly defined the criteria, until we in psychiatry show our usefulness to you, and until you who are in general medicine verbalize your criticisms, your fears and your frustrations about us.

The psychiatrist should venture to criticize only if he believes he can improve and clarify. The purpose of this paper is to indicate, on the basis of condition, those patients who should be considered for psychiatric referral. The criteria for referral from a pediatric service to a psychiatric service can be considered in four large categories: (1) diagnostic assistance, (2) management, (3) specific psychiatric treatment, and (4) prevention. What is hoped for, in such a definition of criteria for referral, is not that the number of referrals to the child psychiatrist will be increased, but that psychiatric interrogation as carried out by the practitioner will be enhanced.

Referral for Diagnostic Assistance

Psychiatric evaluation has something to offer early in the consideration of many diagnostic problems. Exemplary is a case in which a twelve-year-old boy recently was dismissed from the hospital. He had been brought to the hospital by the family because of fever and abdominal pain that had been present intermittently for six years, and which had been worse since exploratory laparotomy performed five months previously. The pain was localized to the site of an even older, previous incision, made for presumed appendicitis. Results of examination objectively were negative,

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yet the boy looked pale and apathetic, and he walked in a stooped position, holding his right side in pain. The picture was further complicated by the occurrence of vomiting and loss of weight. A roentgenogram of the small bowel disclosed nothing abnormal. The boy's general appearance, the vomiting and loss of weight and the presence of fever suggested physical disease. The long history in which nothing abnormal had been found, the bizarre gait and the negative results of abdominal examination bespoke emotional conflicts. This was a situation in which pediatric referral to psychiatry was indicated for purposes of diagnosis. Yet parental acceptance was made possible only by a very direct, authoritarian insistence on its need.

In this case the boy's complaint of abdominal pain was, in fact, unusual sensitivity at the site of the first surgical incision. Detailed psychiatric interviewing permitted correlation of the surgical procedure with unusual anxiety about punishment for angry thoughts of the boy toward his father. The pain did not have the characteristic pattern of the usual syndromes of organic pain, yet the vomiting, loss of weight and presumed fever, later disproved, placed the onus of decision as to what to do on the physician who had applied the "functional" label. Continued interviewing made it possible to correlate the appearance of abdominal pain with fear of separation from the overindulgent mother, who incidentally in her anxiety misreported the presence of fever.

This kind of diagnostic dilemma is not unusual. The easy course in this case would have been to perform a third laparotomy. The constructive and medically sounder approach was that of using to the fullest extent the special technic of psychiatry. Thus, money which would have been spent on surgery could be turned into psychiatric treatment; the anxiety and discomfort of another surgical procedure were not added to the existing neurotic problem and the direction of necessary therapy was clarified.

In many cases syncope, fatigue and weakness must be regarded in a similar manner. If any of these are presenting complaints, and if the history does not establish the usual physical causes for these symptoms, psychiatric referral should be considered. The case mentioned at the opening of this paper is an example. For eight years the boy had been a chronic invalid. With each new diagnosis or treatment program everyone's anxiety increased, including that of the physician. With each lack of improvement everyone became more discouraged. Only after eight long years, with absences totaling two and one-half years of schooling, untold loss of physical and social interchange and continuing conflictual anxiety, was the complaint of fatigue treated as a psychiatric symptom. In this instance, in spite of the extraordinary delay in psychiatric referral, a program of intensive treatment permitted the child to improve sufficiently to return to school.

The problem of headache is slightly different, for there is increasing evidence that children with migraine, as well as those with so-called tension or vascular headaches, should be considered for psychiatric referral. Although there may be vasomotor, migrainous, "allergic" and other sometimes ill-defined factors, the role of emotional problems always should be evaluated. If headache is not related to an expanding intracranial lesion, an effort should be made to define the nonverbalized anger of which headache may be the somatic representation.

The problem of movement disorders currently is an enigma. Both neurologists and psychiatrists recognize this group of conditions as being within the hinterland of the two specialties. If the movement disorder is compatible with the diagnosis of tics or compulsive rituals, psychiatric referral, with careful evaluation of parents and child, is indicated. If the story is that of a bizarre convulsive disorder or one not responding to the usual anticonvulsive medication, the same is true. Proper diagnosis and treatment are possible only if psychiatric factors have been weighed as part of the diagnostic evaluation.

In summary, then, referrals for diagnostic assistance should include:

1. Unusual instances of abdominal pain, particularly if they are associated with bizarre behavior.

2. Headache problems, including migraine, if results of neurologic examination are objectively negative.

3. Syncopal attacks, especially if there is a history of physical brutality in the family.

4. Movement disorders, and particularly instances of multiple tics, pseudoepilepsy or pseudonarcolepsy.

5. Problems of fatigue and weakness, if results of routine laboratory studies are negative.

Referral for Management

Referrals for management include instances of recognized psychiatric import frequently not referred because of a number of odd excuses—their excessive number, the unavailability of psychiatrists, the cost, the family's bias against psychiatry, or, I am sad to say, the assumption that a psychiatrist will only add confusion.

Many are the patients who should be referred. For instance, some children are obese because of parental fostering of their pattern of overeating. Psychiatric evaluation of the parental background may clarify for everyone the use of food as an inordinate symbol of affection, with more satisfactory dietary programming for parents as well as for the child. Diet definitely may be indicated, but so also is planning for increased family activities. On the other hand, some obese children present more disciplinary problems than do children who are affection hunters. When such children are involved, psychiatric evaluation can alert everyone to the need for more consistent discipline; the dietary regimen should come at a later date, since experience shows us it will not be effective until limits on behavior have been strengthened. The same holds among children with anorexia nervosa. Treatment alternatively may be best focused on diet or on emotional conflicts.

Similarly, the management of patients with severe primary dysmenorrhea will be ineffective until family patterns are understood. Recently, we interviewed a fifteen-year-old girl with incapacitating dysmenorrhea requiring forty-eight-hour absences from school, monthly. For four generations the women of this family had had severe dysmenorrhea until the first pregnancy, and had been treated by presacral neurectomy, or the use of a pessary, or cervical dilatation, or deep roentgen rays or narcotic agents. Fundamentally, the problem was one of a continuing conflict between mother and daughter, resulting in highly mixed feelings about the acquisition of the feminine role, of which menstruation is the most colorful symbol. Psychiatric referral for purposes of management permitted proper interdigitation of medication and psychotherapy.

So it is also with unexplained urinary incontinence and with intractable asthma. These are conditions apparently caused by multiple factors; empirically it should be assumed that the failure of the patients to respond to the usual treatment procedures should raise the question of psychiatric evaluation as a step in better management.

In problems of intersex and premature development the psychiatric evaluation should be part and parcel of the routine evaluation, since by the very nature of the physical factors there is in these problems psychiatric tinder already ablaze with conflicts.

The psychiatric problems presented by gunshot wounds, amputations and chronic disfiguring illnesses are ones which eventually will become the treatment responsibility of the practitioner. In the present stage of medical interchange, however, all too few practitioners have become aware of the mourning reaction, the body-image problems and the anger which always accompanies these conditions.

As one little girl said to me, "I know that surgeon did a good job, but I sure hate his guts for taking my arm away."

We know now that not only ineffective rehabilitation but also much of the postoperative pain of such conditions is related to suppressed anger. Few practitioners are motivated to deal with this aspect of treatment. Psychiatric referral should, therefore, be part of the general management program.

Physicians in general are alert to the characteristics of hysterical paralysis, and effectively treat the condition without unusual use of medication or undue fostering of invalidism. The natural course of the disease is such that the rate of symptomatic spontaneous recovery is high. One is left, then, with the problem of selecting those patients who require immediate psychiatric treatment and those best treated by the tincture of time. Herein lies a service which the child psychiatrist can offer.

The problem of mental retardation is in much the same province. Practitioners evaluate with amazing accuracy the intellectual level of children without the help of psychologic tests. There is, however, a significantly large group of children who function at a retarded level because of severe anxiety and who need specific psychiatric treatment as well as a management program.

Similarly, the schizophrenic child should have the combined counsel of practitioner and psychiatrist for proper management. With some basis for the hope that hospital facilities for psychiatrically disturbed children in Minnesota will be increased, early diagnosis and outlining of management in the field indicated become imperative.

In summary, then, children should be considered for referral for management purposes if they have: (1) obesity or anorexia nervosa, (2) incontinence or dysmenorrhea, (3) intractable asthma, (4) endocrine sexual problems, (5) physically disfiguring conditions, (6) hysterical paralysis, (7) mental retardation, or (8) schizophrenia.

Referral for Treatment

Many conditions are recognized as falling entirely within the psychiatric realm, but even so, referral frequently is delayed because of the presumed difficulty of treatment, or the distance involved, or the lack of hospital facilities. These realistic matters will be overcome only if we diagnose, refer and treat as correctly as possible; only if realistic pressure is continually placed on the psychiatrist will the specialty and its facilities expand adequately.

For many years, referral of the acutely psychotic patient was considered a necessary evil. With the increase of knowledge concerning proper treatment, referral now has become an emergency. Resolution of psychotic symptoms now is possible, provided the child and parents are seen quickly, before there is loss of clues by which effective resolution is possible.

The treatment of the delinquent child also has been refined. Rare is the time when we do not have such children and their parents in therapy. It is a long and arduous task for all concerned, but it is also highly rewarding if success is attained. Carefully evaluated psychiatrically, those delinquent children whose problems seem more hopeful of solution should be selected for treatment. Typical are two patients recently dismissed from therapy. The boy, now sixteen years old, was referred because of possession and sale of drugs; the girl, now fifteen years old, for truancy and sexual misbehavior. The delinquent child should be referred for treatment evaluation, and should not be dismissed as a constitutional psychopath.

The child more than four years old who soils himself or wets the bed, the child who misses school because of poorly explained physical symptoms or severe anxiety and the youngster with attacks of hyperventilation frequently are viewed as "going through a stage"—and so they are, but it is a stage of neurotic illness. They may overcome these symptoms but acquire others more severe if psychiatric treatment is not provided. The apparently spontaneous abatement of symptoms is deceptive, and should not be confused with cure.

Typical is a girl of nine years, referred for encopresis of five months' duration. Examination revealed a child functioning on the level of a five-year-old, unconsciously smoldering with jealousy toward her two younger siblings, withdrawing from playmates and viciously expressing her angry defiance by the pattern of fecal soiling. Interrogation of the parents about their backgrounds and the background of the child in play sessions revealed the conflicts for which successful therapy should be possible. Six weeks of treatment now should free the girl both of symptoms and of the need to remain immature. Once freed up emotionally, she can take advantage of the natural developments in living which push toward independence and growth.

Another group of children in need of immediate short-term therapy are those who exhibit infantile behavior after surgical treatment or trauma.

Typical is a thirteen-year-old boy operated upon for cerebellar tumor. Postoperatively he was mute, unable to care for himself in bed in spite of the fact that results of a neurologic examination were essentially negative. Observation revealed a hovering, ever-present, obese, well-meaning but far too indulgent nurse-mother who was conveying to her son the unconscious wish that he remain invalided and incapable. The psychiatrist insisted that the mother have no extra visiting hours, and that she work part time at another hospital. The boy was told very directly about what we considered the problem to be, and within seventy-two hours he was discussing his awareness that his mother had infantilized him. It is probable that some spontaneous adjustment would have taken place slowly, but psychiatric intervention speeded up the process and permitted all concerned to discuss their feelings and attitudes.

One could continue with untold examples. Suffice it to say, in summary of this part of the paper, that children who are delinquent or are afflicted with encopresis and enuresis, acute anxiety and problems in discipline, school phobia, attacks of hyperventilation, sexual aberration, acute regression, depression, obsessive compulsive neurosis or acute psychosis should be referred routinely for psychiatric treatment evaluation.

Referral for Prevention

There is another group of criteria for referral, best labeled "preventative." The child who is unusually modest or unusually destructive in the office, or one who cannot be separated from his parents, represents a case in point. Psychiatric symptoms in themselves may not be present, but family difficulties certainly are. The *insistence* of the mother on the presence of some seriously morbid condition in the youngster constitutes another instance in which pediatric referral to psychiatry should be considered. Such mothers have a strong, unconscious destructive wish toward their children which may actually destroy, either directly or by enforced invalidism. Of equal import are the family patterns of brutality, sexual promiscuity, delinquency, excessive immodesty or unusual disciplinary vacillation. These are behavioral patterns which reflect neurotic maladjustment within the family, and they cannot fail to influence the development and welfare of the child. The time is at hand when practitioners should offer pediatric referral to psychiatry for evaluation of the effects of such behavior, just as they would evaluate the contacts of a tuberculous patient.

A word should be said about the use of drugs. It is a sensible rule-of-thumb that a child who needs sedatives or tranquilizers is one who needs psychiatric referral. In the absence of definitive help, medication is a necessary evil, but it should be a temporary expedient only.

Summary

Pediatric referral to psychiatry should have a fourfold goal, which would include diagnosis, management, therapy and prevention. The basic requirement for referral is that of the practitioner's sensitivity to the importance of emotional conflicts in disease. The psychiatrist must enhance such sensitivity by making himself more easily available and by increasing his efforts to be of practical and immediate assistance.

PROBLEMS OF SELECTION IN STUDIES BASED UPON HOSPITAL AND AUTOPSY DATA

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burden of proof as to the non-existence of important selection forces lies with the reporting investigator. It is he who must present a convincing case. Data which may be given one of two opposite interpretations, depending upon the existence or non-existence of selection forces which are not even investigated, must be considered of questionable value.

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The Physician as Scholar and Humanist

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FOR MANY years I have observed the fine creative output of your school in medical science and medical education. It was, therefore a great pleasure to be given this opportunity to close the geographic gap and visit you in person, though the pleasure was somewhat neutralized by an anxious search for a justifying message. Timothy Dexter, a famous character of Newburyport, Massachusetts, once sent a shipload of coal to Newcastle on the advice of a humorous acquaintance. The coal arrived during a miners' strike, and the voyage was unexpectedly profitable. As I bring my coal to this Newcastle, I can hope for no such fortunate scarcity.

Since this Convocation is, in part, a welcome to the incoming class of medical students, it is not inappropriate to congratulate the newcomers on their choice of a profession. And to explain why I, after a long and varied experience, can say with undiminished enthusiasm that medicine is the best lifework of all. No other profession has such a broad spectrum of opportunity, for it presents two (I am tempted to be dogmatic and say *the* two) most exciting human potentials: (1) the gratification of man's curiosity, his urge to know and to understand the phenomena of nature; (2) the desire to be of service to his fellow man—on one hand, the pure scientist widening the periphery of our knowledge; on the other, the family physician dispensing healing and comfort. The extraordinary quality of our profession, however, lies in the fact that one is not forced to confine himself to either end of the spectrum, for a variety of combinations of the investigator and the healer is not only possible but usual, and, indeed, both elements are necessary for the best practice of medicine. This involvement of the whole spectrum is particularly evident in a medical school where laboratories and wards are side by side not only geographically but, at long last, intellectually. Time and interest can be divided between the two areas in many different proportions according to taste and opportunity.

Furthermore, the physician can be a teacher. Teaching is the shortest route to learning. The physician can concern himself with community problems as a public health officer, and the industrial specialist plays a significant role in economic affairs. If an interest in administration appears, the physician may head a hospital, become a city or state official or be concerned with a health insurance program.

On the material side, it is recognized that medicine is the most stable occupation in almost every part of the world, for social, economic and political upheavals usually leave the physician relatively untouched. During the great depression of the thirties his budget was cut, but he suffered no intolerable idleness, and he had incredible opportunities for the generous gift of his services. But I hasten to add that any young man or woman who enters the study of medicine with a primary interest in its financial rewards is doomed to disappointment. The scientist is never adequately recompensed, and the practitioner works very hard for a comfortable living. As Christian found in Pilgrim's Progress:

> "They came on a delicate plain, called Ease, where they went with much content, but that plain was but narrow so they went quickly over it. Now on the further side of that plain was a little hill called Lucre and in that hill a silver mine; which some . . . had turned aside to see . . . and could not, to their dying day, be their own men again."

The keynote of the multi-faceted profession of medicine is dedication; with the loving and earnest devotion which this implies, no life offers more joy and satisfaction.

The envy that I have had each year for the young student about to cross the threshold of medicine for the first time has in recent years been tempered by the realization that medical science and medical education have been offering all of us many new thresholds. This is a period of greater ferment

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than any since Abraham Flexner's famous report acted as catalyst. Curricula are being scrutinized and extensively revised, new disciplines created and old ones discarded, the content and perimeters of today's traditional departments would be almost unrecognizable by a freshly graduated student of 1910. There have been striking changes in pedagogical techniques necessitated by the tremendous advances in the scientific world. A slow, and not unsatisfactory, evolution in these techniques for producing a physician has taken place by the wholly appropriate use of the experimental method. Yet there is a current demand for greater velocity in this evolution.

This urgency is the normal result primarily of healthy and impatient dissatisfaction with things as they are. But it is encouraged to a certain extent (and understandably so) by the dispensers of funds, both public and private. These agencies usually prefer to back a relatively new departure than to enrich a pre-existing, even though successful, project—a new program, rather than an old institution. Such support may induce an emotional commitment which might tend to limit the validity of the experiment. But in addition, it may stimulate in this fertile soil of change, tangential and spectacular alterations a little beyond the spirit of the experimental method, and too often oriented to the hobbies prevalent in the source of financial support. Though it is better to go too fast than to stand still, the greater the rate of advance, the greater the necessity for a thoughtful examination of our goals.

The first goal which I wish to discuss is our responsibility to the undergraduate medical student; the second, less easy of specific definition, is essentially the role of the internist as teacher and practitioner.

For simplification (and I am afraid that I shall be guilty of continual oversimplification), one may divide the pedagogic procedures of medical school into education by osmosis depending on exposure to a proper environment, education by appropriately stern discipline, education by active training of the mind while imparting information and, finally, development of an appreciation of the philosophy of science through participation in research. A further function of medical school (and in some respects its highest function) is the discovery and stimulation of talent. Creation of an eager curiosity about man and nature is a sure guarantee of the students' continuing growth and there is no greater contribution to the advance of medicine than the cultivation of an original and insatiable mind.

The goals of training the mind and participation in research will receive my main emphasis, but a brief amplification of the other points is necessary for their dismissal. A student's observation of his environment is the strongest force for fostering his capacities for tact, compassion and a sense of responsibility. He will respect, ethically, as well as scientifically, those values which his faculty respects. The finer human qualities so important in a physician are largely inborn, but they can be suppressed or they can be stimulated by the general climate of a medical school.

The grave responsibilities of patient-care require the strictest self-discipline, and the undergraduate period should initiate this type of behavior. While example plays a large role, active external discipline is a valuable adjunct. The student should realize, for example, that, regardless of his academic standing he will be summarily expelled if he inexcusably neglects his duties to a patient assigned to his care. However, there is strong support for the opinion that premature deputation of such clinical responsibility, as is done in certain home-care programs, is an unwise approach.

It is in no derogation of the important functions that I have just mentioned to state that the basic goal of a medical school is the production of scientists and, when possible, scholars. The outstanding characteristic of a scientist in any field is disciplined intellectual power; the power of critical observation and sustained attention, the power of analysis uncluttered by irrelevancies, the power to organize and express ideas and the imagination to see their possibilities. Granting that some of these qualities also are more or less inborn, the major portion can be evoked and developed when proper leadership is afforded.

"Scholar" is a more generic term applicable to the humanities as well as to the sciences. The dictionary is disappointing so I beg your tolerance for an improvisation. A scholar is one who has not only an understanding of his discipline in depth but is preoccupied with an eager pursuit of even more profound understanding: he is not satisfied with his solid foundation of knowledge, he has a divine urge to search further.

In medical school we should strive to *produce* medical scientists and to *discover* medical scholars.

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The progressive enlightenment of medicine depends upon the physician as scholar, the sound practice of medicine upon the physician as scientist.

Although none would deny that the imparting of factual knowledge is a major objective, yet those of us who entered medical school almost fifty years ago have observed a steadily declining emphasis on lifeless facts *per se* and an increasing concern with the mechanisms of disease. Furthermore, there has also been a decreased emphasis on the training of specialized skills, for it is generally accepted that these should be perfected, in main, during the graduate residency periods. However, a competent teacher finds no mutual exclusiveness between the communication of information and the development of scientific critique. But the continued professional growth of a physician depends on the way he has learned to use his mind rather than on the facts that he was able to store in it during his undergraduate years. Science transforms the static into the dynamic and adds the delightful savour of understanding. The joy of understanding is both the closest rival of the joy of service and its most effective partner. Francis Bacon was aware of this emotion when he wrote 350 years ago, "For all knowledge and wonder (which is the seed of knowledge) is an impression of pleasure itself."

The fundamental requirement for training the student to be a scientist is an atmosphere of research. In every department he should find clear evidence of a driving intellectual curiosity and, in addition, he should be told the exciting story of significant scientific advances so that he may experience vicariously the pain and pleasure of exploring the unknown. He will thus derive a familiarity with the type of logical thinking and the rigid quality of experimentation that have been successful in revealing new facts and their relationships. The amenability of the basic sciences to such an approach is obvious, but history-taking lends itself equally well to introducing scientific critique. An analysis of the multiple components that appear in the course of a clinical appraisal presents an excellent opportunity for exploring the principles of logic.

However, no pedagogic brilliance can equal the value of personal participation in a research problem. Each academic year should provide free time, and adequate space should be available to the interested student. The faculty can arouse this interest and point out the high practical value of research experience to the continuing development of a physician. It is my opinion that time used in this manner will lay a more effective foundation for the practice of medicine than equivalent time spent visiting patients in their homes or acting as apprentice to a general practitioner.

Norman Campbell writing on "Science, Imagination and Art" in a recent number of *Science* makes this inspiring comment: "There are degrees of scientific as well as artistic imagination. But the least of us can share in some small measure these achievements. A man need not abandon all pretentions to the proved title of artist because he could not design the Parthenon or write the Fifth Symphony. Most of us who have attempted to advance science have had our all too brief and passing moments of inspiration; we have added a single brick to the mighty structure or finished some corner which the master in his impetuosity has overlooked. And though our tiny efforts rightly pass almost unnoticed by the rest of mankind, they have a value for ourselves beyond what we can tell; one instant we have stood with the great ones of the earth and shared their glory."

I recently spent an evening with a small group of our Columbia students talking informally about the value of research. A recurrent question was, "How do we go about it?" May I pass on to the incoming class the practical suggestions that arose therefrom? A small number of students have unusual talents or special experience which lead them at once to an appropriate opportunity; they are fortunate. Among those less clearly oriented, will be a few who enjoy and are competent in a definite technical approach, such as chemistry or microbiology. They should seek an investigator using this approach and attach themselves to him. Individuals with no special interest or experience, yet with a desire to participate, may choose a member of the faculty whom they find inspiring and follow his intellectual leadership.

For those disliking any form of laboratory work, there remain at least two more avenues of research. First, is clinical study. While much scrutiny has been given to the natural history of disease, there are still many gaps in our understanding. The student may not fill one of these gaps successfully, yet probing deeply into any subject is a direct road to scholarship. Another fascinating avenue is found in an examination of the history of some major advance in medicine, searching the library for original papers, trying to discover work as yet unnoticed.

Both clinical and historical studies may give the student an appreciation of the difficulties faced by an investigator, the rigid critique required for evaluation and the remarkable range of man's imagination. A further fruit of such studies might be an idea that would stimulate experimental exploration. To quote Norman Campbell again, "The scientific imagination . . . must come from direct and intimate contact over the widest possible range with the great original works which represent its noblest expression." Finally, I cannot emphasize too strongly the tremendous value derived from writing up the results of such investigations with careful critique by a member of the faculty, not necessarily with publication in mind but for the discipline involved.

Before turning to a consideration of the internist, I wish to comment on two myths that crop up repeatedly as one seeks to improve the scientific quality of medical education and medical practice. They are so absurd that it strains one's credulity to believe that they are sincere responses. These myths are the "cold-blooded scientist" and the "ivory tower." As to the icy intellectual, I have observed many scientifically trained clinicians and, also, men less fortunate in this respect, and, as I have stated on other occasions, I have never seen a warm, sympathetic person frozen by a research career or a cold, tactless individual thawed by general practice.

The "ivory tower" is more subtle, less clearly definable. Its usage seems to imply, not the widened perspective of a high elevation but isolation from earthy affairs, grudgingly conceded to be located in a durable and lovely encasement of ivory. The whole concept represents a last phase of the losing struggle of the physician as artisan against the growing influence of the physician as scientist, best exemplified in full-time academic medicine. The medical artisans seem tragically unaware that most of the recent remarkable advances they employ in the practical everyday care of a patient have come from "ivory towers." Moreover, anyone familiar with the life of a full-time clinical professor realizes that one of his chief problems is to garner enough time from purely clinical duties to provide adequate periods in his none-too-isolated tower. Indeed, a serious threat to the advance of medicine today may be found in the widespread pressure on the clinical faculty to give up strict full-time status and support themselves or their departments by engaging in private practice. Though this tendency is understandable in a period of rising costs, it is a deplorable retrogression.

But there are some criticisms of modern medicine that have more substance. Peter Drucker, writing philosophically in *Harper's Magazine,* suggests that medicine is advancing so fast that it is rapidly "becoming unteachable." He says, "Virtually every experienced teacher of medicine I know wonders whether the young medical school graduate of today . . . is as well taught and as well prepared as his much more ignorant predecessor of thirty years ago." He continues: "The reason is simple. Medical schools are still organized around the idea of disciplines as static bundles of knowledge. But, where a hundred years ago there were at least six or seven such 'bundles,' there are perhaps fifty today. Each has become . . . a full blown 'science' which takes a lifetime to master."

I cannot imagine where Drucker found the so-called "experienced teachers of medicine" who were in such error about the comparative preparation of today's graduate and those of thirty years ago. But, as is usually the case with any criticism, there is a germ of truth. It is true that unavoidable scientific splintering is a handicap to balanced medical education, and there is general expression of anxiety that the sick person is overlooked in the swarm of specialists.

These serious problems can not be resolved by curbing the trend toward specialization, for only through many routes can the biological complexity of man and his diseases be approached effectively. When even the basic sciences are increasingly subdivided, specialization in the clinical sciences involving the total individual appears even more inevitable, indeed is irrevocably accepted. Granting that he possesses an encyclopedic mind, the practitioner so endowed would lack technical skills which only long experience can perfect. It is banal but convincing to assert that no one would wish to be bronchoscoped by a psychiatrist.

The genuine hazards of specialization and our concern for the whole person are parts of an identical problem. It is my admittedly biased opinion that the answer lies in the integrating role of the internist. (I include the pediatrician, of course). The inaccurate term, internal medicine, designates a remarkable discipline. Its active preoccupation with the patient as a person and its exceptional concern with biological mechanisms in health and disease make it the logical candidate for the keystone of the medical arch both in education and in practice. In fact, the arch is already completed in many medical schools and, through group practice, in some communities.

Internal medicine came of age with the establishment of full-time clinical departments. When I served my medical clerkship at Johns Hopkins in 1914, I saw little awareness of the patient beyond his physical signs which were obtained with meaningless virtuosity. I well remember the elegance of wrist motion in percussion that was my unattained model. The chief goal was to predict the gross anatomy of the autopsy. The anatomical pathologist reigned supreme.

Contrast this picture with a modern medical clerkship where the patient is studied as a person, his social, economic and emotional characteristics held as important as his physical status which, in turn, is precisely evaulated by accurate techniques; specialists visit in consultation, their contributions assembled by the internist, and all the data finally developed into an individual pattern. It is difficult to be certain as to the dates or the forces that produced this mature and inclusive discipline which we call internal medicine. However, if I may be permitted the personal testimony of one who has had a ringside seat (with an occasional round of my own) I shall assert again that it is another of the wonderful results of the direct exposure of clinical problems to the full-time medical scholar.

In the first ten years after academic medicine was instituted at Columbia and the Presbyterian Hospital, several steps were taken which gave evidence of an appreciation concerning the important role of the patient's personality. All these steps were supported, happily, and a few were originated by those of us who were called "laboratory men" (with no intent to praise). In the early 20's arrangements were made with the professor of psychiatry for routine instruction of the clinical clerks (on medicine) concerning the emotional problems of their patients. Shortly thereafter psychiatrists assigned to the Department of Medicine began to study the influence of personality on disease. Out of these studies arose the stimulus that led Flanders Dunbar to be Editor-in-Chief of *Psychosomatic Medicine* and one of our internists to represent his field on the editorial board. At about the same time, George Draper, talking and writing of the various "panels" of a patient appraisal, added the panels "personality" and "constitution," an original and pioneering concept which thirty years later was given the term, "comprehensive medicine."

The exposure of the patient to the full-time scholar's critical imagination inevitably led to a recognition that the patient's personality was of equal and often greater significance than the disturbed physiology. Parenthetically, I would suggest that this thoughtful recognition of the broad area of integrative responsibility has been appropriated for crusading purposes under the banner "comprehensive medicine" with the emotional overemphasis typical of such a reaction.

Our appreciation of the total individual was greatly enhanced by the accurate and easily available quantitative techniques developed in the research laboratories, many of them in clinical departments. As a surprisingly complete physical appraisal became possible, the gaps in understanding due to psychological or environmental influences could be evaluated more clearly, and the etiological role of the emotions was less a matter of guesswork. So, gradually, as a result of comprehensive understanding (and I prefer to use the term in that association), the humanistic instincts characteristic of the healer from time immemorial were afforded enlightened and effective usefulness. The prompt impact of this development on medical education was guided largely by internal medicine.

As undergraduate medical education has concentrated more on training the mind and less on indoctrination of skills, a rather profound reorientation of curricular emphasis is in progress. Time spent on learning static facts, such as the origin and insertion of muscles, is devoted to the biology of the cell, physiology is concerned more with human reactions than those of the frog, and pathology emphasizes the dynamics of disease. But the *most* important development has been a general unifying tendency with co-operation between all departments, joint seminars and clinics, and interchange of faculty members. As departmental walls crumble (I speak only of pedagogical walls) and common educational goals appear, the integrating potential of internal medicine invests it with the leading role in an evolving medical school. All clinical departments now have active roots in the basic sciences, but the deeper and more widespread ones of medicine and its intimate contact with all the other clinical branches gives exceptional insight to the medical man with corresponding educational perspective. I hope that this presentation of the functions of the internist has not appeared to be an arrogant exaggeration; such functions seem to me a natural corollary to the broad role of an integrator. Furthermore, the internist is more often privileged to dig deeply into some area—one qualification for a scholar.

The physician has been a healer and often a humanist for centuries. He has fought unarmed by knowledge and hobbled by tradition. Though he brought comfort to the mind, often he did harm to the body. The leading dictionary definition of "heal" is "to make whole." At last we are able to touch that goal. Here again the helm is usually assigned to the internist because he is better trained as a pilot than the rest of the crew. Though he may lack the technical skills to fix the radar, repair the generator or adjust the diesels, he must understand the priciples under which they operate. It is a complicated and exciting responsibility.

The internist, thus, is cast as the family physician, the keystone of the clinical arch. The family doctor of fifty years ago was fortunate in knowing his patients before they consulted him, his approach was intuitive, based on general familiarity with his neighbors. A well-trained family physician of today can take histories that are more illuminating as to his patient's personality and environment than were they to be his next-door neighbors. Moreover, the investigation is guided by a technical knowledge of psychology and an understanding of emotional influences on health. Sooner or later, no facet of the patients' lives will have failed of scrutiny. This procedure is not only more sophisticated but more effective than an intuitive assembly of gossip. The modern internist can give as much warmth and compassion as his predecessors, but he can add to these important gifts an extraordinarily detailed scientific appraisal. In no sense do I discount the value of intuition, for the complexity of modern medicine makes even greater demands on this rare capacity for the unconscious analysis of experience.

A humanist has been defined as "a student of human nature or of matters of human interest; one versed in human affairs and relations." Considering the physician as humanist, it is necessary to extend these qualifications, for the physician as humanist requires also a deep affection for mankind and a profound respect for the dignity of man. This is something beyond a warm friendly attitude toward patients. The application of this philosophy to a single individual seeking help needs little discussion for no other member of a community is afforded the variety of opportunities for service that fall to the lot of the physician. No one else is given as honest or as complete a picture of the individual. The doctor must be "a student of human nature," and his experience guarantees that he will be "versed in human affairs." By our dictionary, therefore, he is a humanist; if he really cares for his patients (and every good doctor does) he has then completely fulfilled our specifications.

The more difficult problem for the physician is the application of his humanistic responses to groups, to the community at large. In strictly medical affairs there can be no argument, he must seek and exercise leadership. But in sociological, political and economic realms, he will find himself torn by conflicting motivations. It would be presumptuous of me to offer advice in this delicate area. The physician represents a learned and respected profession, he is a citizen, and he is close to the hearts of many of his fellow-citizens. His consequent influence may be enormous, and his conscience must tell him how and when to utilize it, for his responsibility is commensurate with his influence.

Finally, we must never forget that the physician's influence and his capacity for using it *wisely* depend on the fusion of the healer and the scientist, of the humanist and the scholar. To internal medicine falls the privilege and responsibility of guiding that fusion.

EFFECTS OF LITTLE STROKES

Abdominal symptoms of little strokes may easily confuse the diagnosis of cerebrovascular accident in the elderly patient, says Dr. Walter C. Alvarez in a discussion of the small apoplexies.

A little stroke can produce such varied symptoms as nausea and vomiting, simulating acute indigestion or Ménière's syndrome; irritability of the stomach; and a "burning" sensation in the abdominal wall. Quite often such a spell will be followed by rapid aging, some loss of memory and ability and a noticeable change in character and personality.—ALVAREZ, W. C.: The abdominal symptoms of little strokes, *Geriatrics,* 12:164 (March) 1957.

Detection and Treatment of Noninvasive Carcinoma of the Cervix

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CONTROL of carcinoma of the cervix is now within the reach of the medical profession. With the introduction of a planned screening program, the danger from invasive carcinoma of the cervix could be largely eliminated. Instead of a salvage of 50 per cent of women with carcinoma of the cervix, which is now the experience in better treatment centers throughout the world, 100 per cent of women so afflicted could be cured by relatively simple measures. This would mean a saving of thousands of lives each year and a release from untold suffering; it also would obviate the expense involved in the radiation or surgical approach currently necessary to control carcinoma of the cervix. It is the purpose of this paper to present a discussion of the cytologic technique that makes the previous statements credible, to consider the interpretation of its results, and to review some of the problems raised in the course of its application.

Cervical Cytology

An understanding of cervical epithelium is essential both in obtaining and interpreting proper diagnostic cytologic smears. It is a well-established fact that the cervical squamous epithelium undergoes cyclic changes during the menstrual cycle. It also continuously sheds cells, as does all similar squamous epithelium. The squamous epithelium of the normal cervix has an orderly pattern from the basement membrane to the surface. The cells resting on the basement membrane are columnar and are distinguished by large nuclei and little cytoplasm. Squamous cells are referred to as superficial or basal depending on their location near the surface or near the basement membrane. As the superficial cells are shed, they are replaced by proliferation of basal cells. The daughter cells are pushed away from the basement membrane and gradually reach the surface. During this process, they acquire more cytoplasm in which keratin granules develop; at the same time, the nucleus becomes smaller and pale.

In the normal smear, both basal and superficial cells may be present, the latter being predominant. These superficial cells are large and angular, with a small central nucleus. They are likely to be wrinkled and folded. The cytoplasm stains faintly and may be either basophilic or eosinophilic. The basal cell varies greatly in size but is somewhat smaller than the superficial cell. In general, it is oval or rounded. The cytoplasm is usually basophilic, and the nucleus is relatively large and granular. In precancerous states, some of the cells shed are not typical of fully developed surface cells.

The difference between atypical benign cells, such as may occur in leukoplakia or in inflammatory hyperplasia, and the malignant cells in carcinoma *in situ* is not always clear. Little is known about the development of carcinoma *in situ* of the cervix. It is even difficult to determine whether growth initially is sluggish, as is the case in carcinoma of the skin. A lag of eight to ten years may occur between the initial appearance of carcinoma *in situ* of the cervix and the time of invasion. In the most typical form of carcinoma *in situ,* malignant changes are present in all the cells of a segment of the squamous epithelium. The cells are not keratinized and are spindle-shaped, with enlargement of the cells of the basal layer. Mitotic figures are generally numerous. Great variation often exists in the size of cells, and some multinucleated cells may be present.

The differences between benign and malignant cells are mainly matters of degree rather than of kind. The nucleus of the malignant cell tends to show alterations in shape, abnormalities of the chromatin and enlargement of the nucleolus; the ratio of the nucleus to the cytoplasm is increased. The cytoplasm is secondary to the nucleus for diagnostic purposes; the important features are the amount of cytoplasm and the shape of the cell.

From the Section of Obstetrics and Gynecology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

Read at the meeting of the Northern Minnesota Medical Association, Hibbing, Minnesota, September 6, 1957.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

The Cervical Smear

There is, at the present time, a confusing multiplicity of techniques for obtaining cervical smears. Only the one in use at the Mayo Clinic will be described here. This is not to say that other techniques are not suitable, but this method has been satisfactory in the experience of my colleagues and me. After exposure of the cervix with a speculum, an Ayre wooden spatula is inserted into the cervix and vigorously rotated. The material on the spatula is then smoothly smeared on a glass slide and immediately fixed in 95 per cent alcohol. Care is taken not to allow drying in air before fixation. The slides are stained with hematoxylin and eosin.

The stained slides are examined microscopically, with the aid of a mechanical stage, by a trained technician, who marks questionable or malignant cells. A pathologist, trained in cytology, studies the marked slides. He also checks some of the unmarked smears to determine whether the specimens are adequate and to spot check the technician scanners. If the slide is improperly prepared, neither the cytologist nor the cytotechnologist can be of much assistance. The accuracy of their report depends on the quality of the material submitted.

The cytologist is, of course, the key figure. He must not only be a pathologist but also must have considerable training and interest in the relatively new discipline of cytology. His right hand is the cytotechnologist, who relieves him of much of the work load. At present, many cytotechnologists are being trained throughout the nation; this training program is in large measure subsidized by the American Cancer Society.

Interpretation and Results of Cervical Screening

Many terms are used to report the findings of a cytologic study. The categories we employ are as follows: group 1—negative; group 2—probably benign; group 3—suspicious; group 4—probably malignant; group 5—malignant.

If a group 3, 4 or 5 report is obtained, the next step always must be cervical biopsy. This may consist of multiple punch biopsies of the squamocolumnar junction or study of tissue removed during cold conization. The diagnosis of carcinoma *in situ* of the cervix must be made from this material. If confirmation is not thus obtained in the presence of frankly positive smears, the wisest course then may be simple hysterectomy or removal of the offending cervical stump so that the completed specimen can be checked by the pathologist. This does not apply when the report indicates only possibly suspicious cells or when the presence of associated pathologic lesions of the cervix may explain the atypical cells in the smear.

During the past few years at the clinic, the cytology program gradually has been expanded as cytologists and cytotechnologists became available, so that soon my colleagues and I hope to be able to offer the test to all women more than twenty-five years of age who are examined at the clinic. From 1948 through 1953, some 30,310 smears were examined, resulting in the eventual diagnosis of 270 cases of carcinoma *in situ* of the cervix in women who had no gynecologic complaints. This is an incidence of 8.9 per 1,000. In repeat smears obtained at a later time on the "negatives" in this same group, the incidence of carcinoma was only 1.7 per 1,000, which is a strong indication of the importance of the screening technique.[1] An occasional false positive result is still obtained but these are much less to be dreaded than the false negative result, which sends a patient on her way falsely reassured about her physical condition.

Treatment

After the diagnosis of carcinoma *in situ* of the cervix is made, the question arises concerning the proper treatment to be instituted. The gynecologic literature is full of varying treatment programs. From their multiplicity, it is obvious that much remains to be learned about this particular lesion. For example, a frankly positive smear substantiated by biopsy in a woman past thirty-five years of age who has had several children means hysterectomy. But what of the recently married nineteen-year-old girl who is anxious to have a family? Is it safe to allow her to have one or two pregnancies under observation with repeated smear and biopsy? The easy course for both physician and patient is immediate hysterectomy. But with a co-operative and stable patient and a conscientious physician, surely some of these girls can be allowed the privilege of motherhood. Nowhere is the physician-patient relationship more important than in such instances.

The treatment of carcinoma *in situ* of the cervix follows three general trends, namely observation, limited therapy or hysterectomy. The last trend far outweighs the first two. Under limited therapy,

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The Use of Continuous Suction-Drainage for a Variety of Surgical Wounds

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SURGICAL wounds with actual or potential dead-space present a distinct problem to the surgeon in that they tend to increase morbidity, prolong hospitalization and favor the development of the wound infection. The problem can be solved in most instances by the use of continuous suction-drainage in such wounds. This method of management is far superior to the use of wick drains, packing or pressure dressings. It is comfortable for the patient and it permits the surgeon to perform a primary closure of wounds, which he might otherwise elect to leave partially or completely open for secondary healing.

We have used continuous suction-drainage in the management of a wide variety of surgical wounds over a period of five years or more, having originally read of its use in radical mastectomy wounds. We found continuous suction-drainage very satisfactory for the management of the wound of a radical mastectomy; the method prevents the accumulation of serum, permits immediate active motion of the extremity on the affected side, and eliminates the need for bulky compression dressings. This technic, together with primary closure, also proved equally satisfactory for other troublesome wounds, such as the perineal wound of an abdomino-perineal rectosigmoidectomy; pilonidal cystectomy; radical neck dissection; herniorrhaphy for huge scrotal hernia; hydrocelectomy, and other wounds with large areas of potential dead-space.

The technic for continuous suction-drainage is quite simple. We use one or two polyethylene tubes, depending upon the size of the wound, sizes 13 to 16 F. Multiple lateral perforations are made in the tubes. The tubes are inserted into the depths of the wound and anchored securely to the skin incision. The wound is completely closed. We often elect to use no dressing except a thin layer of cotton which is stuck to the incision with an ether-benzoin solution. This dressing eliminates the need for adhesive tape and bulky, restrictive dressings.

Continuous suction is applied to the polyethylene tube in the postanesthetic recovery room and continued for forty-eight to seventy-two hours, or more, as indicated. Any type of nasogastric suction machine that supplies continuous suction is satisfactory for the wound suction. The suction is discontinued temporarily whenever necessary to permit ambulation of the patient (full activity on the part of the patient is encouraged). A simple dry dressing is applied over the site of entrance of the suction tube when it is removed, but the cotton-benzoin dressing is left in place until one is ready to remove the skin sutures.

We also use this same type of simple continuous suction-drainage within the abdomen or deep cavities when excessive drainage is expected. For example, the continuous suction technic is useful in draining a raw, denuded pelvic cavity; a pancreatic stump or an ulcer bed deep in the pancreas; an indurated duodenal stump which is not as securely closed as usual or a uretero-pelvic or ureteral plastic procedure when excessive leakage of urine is anticipated.

When this technic is used for intra-abdominal or deep cavity drainage, we often encase the polyethylene tube within a rubber wick drain, making, in effect, a modified Babcock sump-drain.

The simple technic described above is advocated for the management of a wide variety of troublesome surgical wounds. It decreases morbidity due to wound complications, permits a primary closure of wounds which otherwise would be left open for secondary healing, and greatly simplifies wound care.

From the Austin Clinic, Austin, Minnesota.

Presented at the Southern Minnesota Medical Association meeting, Lake City, September 9, 1957.

Case Presentations

Carcinoma of the Stomach Complicated by Pregnancy

Report of an Unusual Case

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THE following case of carcinoma of the stomach complicated by pregnancy is of particular interest.

The pertinent history begins at the Mayo Clinic in 1947 when the patient was twenty-seven years of age. She complained of epigastric distress that had been present for about one year. The patient presented a nonspecific type of history but had noted that the distress was associated with occasional nocturnal vomiting. Results of general physical examination and laboratory examinations, including x-ray examinations of the gall bladder and stomach, were within normal limits. The only abnormal finding was absence of free hydrochloric acid in the gastric content following a test meal. No evidence of organic disease was discovered, and a trial of dilute hydrochloric acid with meals was suggested.

In September, 1952, at her home in Lake City, the patient complained of gaseous distress, belching and rather severe cramplike pains in the epigastrium with associated vomiting, which often awakened her at 5:00 a.m. Relief was obtained by drinking milk. Roentgenograms of the gall bladder disclosed normal conditions but gastric roentgenograms showed a definite deformity in what was interpreted to be the duodenum. The patient improved promptly on a modified Sippy regimen. In December, 1952, she was found to be pregnant, her last menstrual period occurring October 23. She continued faithfully to follow her ulcer diet and remained asymptomatic until about the last week in March, 1953. At this time severe pain localized to the left side of the back developed. She was hospitalized at Lake City from March 30 to April 2, during which time the pain was controlled by narcotics. It soon subsided and she was released only to return to the hospital in twenty-four hours because of recurrence of the pain with nausea and episodes of vomiting. Because of moderately severe anemia she was given 500 cc. of whole blood on April 15. Analysis of the stools revealed a trace of blood. On April 15 also, the patient noted a recurrence of mild, dull boring pain in the epigastrium. The pain was similar to the pain of the ulcer. Because of the continued severe pain, this patient was transferred to the Mayo Clinic on April 16.

One observer at the clinic noted that the pain seemed to occur in attacks, four of which had occurred since April 1. The pain was located in the region of the left flank, and narcotics were always needed for relief. A complete review of the systems was entirely negative. There had been no chills, fever or urinary symptoms. There was mild tenderness in the middle of the back slightly to the left of the spinal column. Findings on pelvic examination were compatible with five and one-half months' pregnancy. Results of the initial laboratory examinations were reported as follows: urinalysis, essentially negative; hemoglobin 10.8 gm. per 100 cc.; erythrocytes 3,650,000; leukocytes 5400; sedimentation rate 47 mm. in one hour by the Westergren method. Concentrations of amylase and of direct and indirect bilirubin were within normal limits.

Roentgenograms of the chest, the lumbar vertebrae and gall bladder showed nothing abnormal. Because of the location of the pain, a lesion of the kidney was suspected. An excretory urogram, however, revealed only mild right hydronephrosis, which was attributed to the pregnancy. The degree of anemia did not seem more than could be attributed to pregnancy.

While in the hospital the temperature curve was within normal limits, but the patient continued to complain bitterly of pain and demanded narcotics for relief. It was noted that she experienced prompt and complete relief of pain as soon as the needle containing narcotics entered the skin. This, together with her behavior pattern, led to the assumption that her pain might be on a functional basis. For this reason, three days after admission to the hospital, the entire problem was discussed rather thoroughly with her, and she was told that she must stop the use of narcotics. To the surprise of the staff she requested no further medication and simultaneously the pain completely disappeared. The proponents of the functional theory were elated at this amazing recovery; nevertheless it was decided that the intestinal tract should be investigated. When roentgenograms of the stomach were taken they revealed an ulcerating obstructing lesion involving the pyloric third of the stomach. It was the opinion of the roentgenologist that this lesion likely represented a neoplasm. At this point, one of us (Walters) was called in consultation and it was his opinion, as well as the medical consultants', that immediate operation was indicated.

Read at the meeting of the Southern Minnesota Medical Association, Lake City, Minnesota, September 9, 1957.

Dr. Walters is in the Section of Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

At operation on April 24, 1953, an extensive ulcerating adenocarcinoma of the stomach was found extending to the serosa but there was no nodal involvement. Two-thirds of the stomach was removed including 2 cm. of the duodenum below the pylorus. It appeared as though the proximal line of resection for the stomach was well beyond the limits of the lesion, but when the pathologist examined the specimen before the operation was completed, involvement of the proximal end of the resected section made it necessary to extend the resection to the cardia on the lesser curvature leaving only the fundus which was free of neoplastic involvement. An anterior Hofmeister-Polya anastomosis was performed. The pathologist reported that the lesion was an ulcerative adenocarcinoma, Grade 4, with a central crater measuring 4 by 4 cm. and a peripheral zone of superficial spread covering 8 by 6 cm.

The postoperative course was uneventful and the fetus remained active. The patient was ready for dismissal from the hospital sixteen days after operation when a massive gastrointestinal hemorrhage with hematemesis and melena followed by shock occurred. Blood was administered. Frequent washing of the stomach with a large-size tube passed into it, using water at 120° F., did not stop the bleeding. Intragastric exploration became necessary and an emergency laparotomy was performed on May 23, 1953. There was diffuse oozing from the structures in the abdomen. So much of the stomach had been resected that it seemed likely that the anastomosis would be inaccessible to inspection through an incision made in the anterior wall of the stomach. Such being the case, jejunostomy was performed and a catheter was pushed through the opening in the distal loop of jejunum about 10 inches below the anastomosis to the stomach, and directed upward through the anastomosis into the stomach. The purpose of this tube was to keep the stomach free of clots and to serve as a route for injecting vasoconstricting and clot-stimulating substances into the lumen of the stomach. The patient was given 2000 cc. of citrated blood before the operation, and 3500 cc. of blood and 500 cc. of 6 per cent solution of pharmadex at the time of operation. After operation she was given 500 cc. of blood, and 1200 cc. of modified human globin. At the time of the operation 72 mg. of vitamin K_1 was given intravenously and after operation vitamin K was given for several days. The intragastric bleeding rapidly diminished.

Two days after operation the patient began to have mild labor pains. In the next few days the fetal sounds disappeared, and eleven days following the emergency laparotomy and jejunostomy she was delivered of a stillborn female fetus.

From then on convalescence was rapid. The patient improved remarkably. The jejunal tube was removed and she began to eat. She was dismissed thirty-one days following the emergency laparotomy and forty-nine days following gastric resection.

This patient was examined every six months in 1954 and 1955 and every year since then and there has been no evidence of recurrence. She was last examined on March 4, 1957, and she had no complaints. Her appetite was excellent and she had gained 34 pounds. Results of physical examination were entirely normal. Roentgenograms of the stomach showed normal conditions and except for a mild hypochromic anemia her general condition was excellent.

Comment

This patient is particularly interesting because although the lesion was of high degree malignancy (Grade 4) and involved the serosa of the stomach there was no glandular involvement. Although symptoms were bizarre she did have abdominal pain, anemia, and gastric achlorhydria. The initial resection which grossly seemed to go well beyond the lesion proved not too sufficient when the specimen was examined microscopically and re-resection of the remaining portion of the stomach had to be performed before closure. The re-resection segment was without malignant involvement. The postoperative course was complicated by an unexpected gastrointestinal hemorrhage on the sixteenth day following gastric resection. There is some likelihood that a nonviable fetus and a fibrinogen deficiency may have been the cause of the hemorrhage. Unfortunately, studies of blood coagulation, prothrombin time, and fibrinogen were not made prior to the unanticipated hemorrhage following gastric resection.

Although bleeding could not be controlled by washing the interior of the stomach with water at 120° F. introduced into the stomach through a nasal tube, it stopped quickly after jejunostomy was performed followed by administration of vitamin K_1 at the time of operation and vitamin K for several days. Following delivery of a dead fetus on May 21, recovery was rapid. This patient is one of only five returned to the operating room by one of us (Walters) because of postoperative intragastric bleeding, which could not be controlled by gastric lavage with water at 120° F. to remove all intragastric clots, and by blood transfusions. In the last two years, in some such cases, intragastric installations of thrombin or dilute adrenal solutions, or both, have been made.

Stevens-Johnson Syndrome

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THE Stevens-Johnson syndrome is a variety of erythema multiforme exudativum. The etiology is in doubt and the efficacy of treatment is by no means established. The syndrome is thought to have an allergic background, the tissues becoming sensitive to the products of necrosis, to drugs, or eventually to many stimuli. The cortisosteroids are reported to "cure" the disease,[1] and this is adduced as further evidence of an allergic basis for the condition. The following case report demonstrates a dramatic response to ACTH, further supporting a hyperallergic origin.

Case Report

M. J., a ten-year-old girl (patient of E. A. Addy, M.D., Gilbert, Minnesota), was admitted to the Virginia Municipal Hospital, November 13, 1955 with fever and a rash. The youngster had been seen at home, by the family physician, several days before admission. She then presented a temperature of 102° F., and other than general malaise, had no special complaints. The physical examination then was negative, except for posterior cervical adenopathy. She was treated conservatively at home, and was seen the following afternoon with no new physical findings.

The following day, the youngster developed a papular rash on the upper thorax, and an "erysipeloid like" erythema on the malar eminences with butterfly distribution. The youngster was hospitalized on the following day. Physical examination on admission to the hospital revealed a very toxic, dehydrated and acutely ill child. The temperature was 105.4° (R). The pulse was rapid and restlessness was marked. There was a generalized, widely spread rash, involving the palms and soles. The mucous membranes of the mouth were ulcerated and a conjunctivitis was present. She was placed on penicillin, ilotycin and parenteral fluids.

Over the next two days her condition became progressively worse, and on November 15 she was seen in consultation. At this time the patient was very toxic and dehydrated. There was a severe conjunctivitis, with pericorneal vascular injection and marked purulent drainage. The lids were edematous and bluish in color. The lips were cherry red. The buccal mucous membrane presented an ulcerated appearance and considerable sloughing was noted. The tongue was coated and ulcerated. There was a brownish coating on the gums and teeth. The tonsils had a greyish exudate and the pharynx was injected and swollen. The posterior lymph nodes were palpable; no other lymphadenopathy was noted. The neck was supple and no spinal rigidity was elicited. The chest was clear. The heart tones were of good quality, the rate rapid, the rhythm regular, and no murmurs were heard. The abdomen was soft; the liver and spleen were not enlarged and no masses were felt. The skin presented a generalized papular-macular-vesicular eruption; large bullae were in evidence on the face, abdomen and buttocks. Slight pressure over the lesions caused superficial peeling of the skin, leaving a violet-red raw surface. Both sides of the face were blanched and broken blebs with mucoid secretion and some crusting were present on the forehead and cheeks. The genital mucous membrane was ulcerated and some crusting was present.

The laboratory findings showed hemoglobin 15.5 grams; white blood count, 9,300 with 52 per cent segmented neutrophils, 24 per cent non-segmented neutrophils, 21 per cent lymphocytes, and 2 per cent monocytes. On two occasions, L.E. cells could not be demonstrated. The urine analysis and serology were negative. The bleeding and clotting time was normal and the platelet count was 100,000. The total protein was 5.2 grams with 3.7 grams of albumin and 1.5 grams of globulin.

On November 15, 1955, the penicillin and ilotycin were discontinued and the following therapy was instituted: ACTH 20 mg. every six hours, I.V. Achromycin 100 mg. every eight hours, I.V. 10 per cent glucose by continuous drip, Vitamin B Complex and Vitamin C, I.V., and Hydrocortone drops and Neomycin ointment to the eyes. The patient remained toxic until November 16 when the temperature dropped to 100.6°(R) and returned to normal on November 19. There was an apparent dramatic response to the ACTH and supportive therapy. The ACTH and Achromycin were continued for ten days with a gradual decrease in dosage. The skin lesions and mucous membrane involvement cleared rapidly and the youngster was discharged, well, on November 28, 1955, fifteen days after admission to the hospital.

Comment

The case presented appears to fit into the condition referred to in the literature as Stevens-Johnson Syndrome. The disease is considered a variety of erythema multiforme exudativum of the "major" or severe type. Because of the variation in skin reaction, involvement of the mu-

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cous membranes, severe opthalmia and the severity of systemic reaction this symptom complex has been described under a variety of names: erythema multiforme exudativum (for the less severe cases), erythema multiforme pluriorificiales,[2] mucocutaneous fever, eruptive fever with stomatitis and opthalmia and Stevens-Johnson syndrome for the more severe cases. Stevens and Johnson[3] described the severe form in 1922.

The Stevens-Johnson syndrome is a disease of the first three decades. It is seldom encountered before the age of three although cases as early as seven months have been reported.

The illness starts out with a fever and general malaise and within three or four days of onset there is a progressive fever, exanthema, oculonasal drainage, laryngeal cough and perhaps some joint stiffness. Three symptoms are characteristic: (1) general malaise, (2) mucosal inflammation, (3) exanthema. Of the general signs, fever is consistently high and oscillating, restlessness and prostration are marked, delirium is often present and rapid dehydration is the rule. A diffuse involvement of the mucosa with severe opthalmia confirms the diagnosis. The buccal and nasal mucosa is always prominently involved,[4] however, the respiratory and digestive mucous membranes may also be involved. Tracheitis and suffocating laryngotracheitis do occur in some cases.[5] Esophagitis, rectosigmoiditis and anusitis have been reported in cases coming to necropsy. The genital mucous membranes are a frequent site of involvement. Conjunctivitis is always present and usually is of the allergic type, presenting a pericorneal vascular injection. Sometimes a purulent conjunctivitis is found, and, in the severe cases, panopthalmitis and ulceration of the cornea is not uncommon with resultant blindness.[6]

The exanthema consists of varied manifestations. Macules, papules and nodules appear early followed by erysipeloid plaques. Vesicles and bullae on a red velvet skin then appear near the original lesions. These bullae burst rapidly and give rise to peeling and ulcerations of the skin. Pressure on the macules causes a superficial peeling of the skin. The bullae are most prominent on the face, limbs and thorax. The eruption evolves through successive stages and lasts two or three weeks unless prolonged by infectious complications.

There usually are very few visceral manifestations. Digestive signs depend on the involvement of the esophageal or rectosigmoid mucosa. Respiratory symptoms are not uncommon and are usually due to mechanical obstruction of the airway by the mucosal lesions. Laryngotracheal suffocation and atelectasis have been reported.[7] An associated hemorrhagic syndrome is a rare complication.

The illness is a self-limited disease, but the length of illness varies widely from ten days in the less severe to several weeks in the severe cases. The temperature remains high, up to 104° to 105° F, and the patient is extremely ill. The skin lesions begin to heal with the fall in temperature. Pigmentation of the skin is common over the healed areas. There is usually extensive peeling of the skin over the affected areas especially on the hands and feet, with the skin coming off in large sheets. The mucous membranes of the mouth are the slowest to heal. It is characteristic for this disease to recur. Several attacks may occur each year for as long as twenty years. The recurrences may be either severe or mild, in the same patient.

The clinical diagnosis is not difficult, but cannot be confirmed by laboratory tests. Blood counts are usually within normal range. Occasionally a leukopenia or a mild leukocytosis is present. The sedimentation rate is often a little accelerated. Cultures of the blood, pharyngeal and cutaneous secretions are negative. Differentiation from eruptive fevers, reactions to antibiotics, dermatitis herpetiformis and pemphigus sometimes causes difficulty. It is not uncommon for these patients to be admitted into contagious disease hospitals as cases of diphtheria, scarlet fever, measles or varicella.[8]

Fatal cases are not common. However Short[7] reported two fatalities in five cases. One of Shorts's cases followed a long course of Thiouracil therapy. It is difficult to obtain mortality figures. In severe cases it may be as high as 10 or 12 per cent.[5]

Since most men consider the Stevens-Johnson Syndrome a self limited disease, no specific therapy is available. This means that supportive therapy is of paramount importance. Fluid and electrolyte replacement require adequate estimation of fluid and electrolyte losses because of high fever and oozing from skin surfaces. Intravenous therapy is essential in severe cases during the first days of illness. Blood and plasma are sometimes necessary in small children to prevent electrolyte imbalance or progressive malnutrition. Nutrition must be maintained by feeding a high caloric, high protein diet with additional vitamins. Cortisone and Corticotropin have been reported to cure the syndrome along with antibiotics to control secondary bacterial invaders. However, since sensitization may play a role, the antibiotics with possible sensitizing properties such as penicillin and sulfonamides should be avoided. Immediate success with intravenous salicylates, along with penicillin and streptomycin has been reported by Bernard and Vincent.[9] Proper local therapy for the skin, mouth, genitalia and eyes is essential to allay discomfort to the patient. The danger of asphyxia may necessitate tracheotomy to relieve tracheobronchial obstruction.

It is difficult to decide if this disease is increasing in frequency or is being diagnosed more often as a result of a greater familiarity with the syndrome. Case reports are appearing in the literature with increasing frequency during the past few years.

Summary

A case report of Stevens-Johnson Syndrome is presented, showing a dramatic response to ACTH. This supports the theory of hyperallergic origin.

The etiology of the syndrome is obscure. However, the lack of a biologic syndrome, the ineffectiveness of antibiotics, alone, and the dramatic effects of corticosteroids seems to indicate an allergic basis for the disease.

The syndrome is characterized by a variation in skin reaction, involvement of the mucous membranes, severe ophthalmia and severe general reaction.

The disease is self-limiting and variable in its course. However, since sensitization may play a role, antihistamines and adrenal steroids are indicated in treatment. Antibiotics except those with possible sensitizing properties should be given to control secondary bacterial invaders. Supportive therapy is essential because of the severity of the disease.

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BURNS OF THE EYELIDS AND EYES

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Continuation Study

Roentgen Diagnosis of Abdominal Tumors in Infants

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EXTRA-ALIMENTARY abdominal masses in infants and children offer a challenge to the pediatrician, surgeon, and radiologist. With reliable clinical and roentgen signs one might establish a diagnosis with enough certainty to decide whether or not to attempt a laparatomy.

The general subject of abdominal masses has not been covered collectively in the roentgen literature, except for Cappuro and Blanco's monograph on topographic localization of abdominal masses in adults. Recently, Nice and Margulis of the University of Minnesota applied this approach in pediatric roentgen diagnosis of abdominal tumors. The case material presented in this course was from their collection.

Physical examination and radiographs in two planes offer the most help in localization of the tumor. Pyelography or barium studies, or both, are the next most useful procedures. Arteriography, retroperitoneal or intraperitoneal air insufflation, or special contrast studies are infrequently necessary.

After the mass is localized, one attempts to differentiate between solid and cystic lesions; the latter are usually intraperitoneal. Solid ones are less sharply demarcated, they will not alter their contonurs during respiration or change in position, nor will they show change in size or shape after aspiration.

One almost must be on the lookout for calcification. Osseous structures or reticulated patterns are found in teratomas and dermoids, and amorphous or stippled calcium may be found in tumor necrosis or in areas of hemorrhage. Hemangiomatous vascular patterns are found occasionally.

The retroperitoneal space is the site of some childhood's most common tumors. They can displace the gastrointestinal tract anteriorly and laterally, the retroperitoneal structures may be displaced, or ureteral obstruction may occur.

Wilm's tumors present as a renal mass with or without intestinal displacement. Collecting system distortion or invasion or both, rather than nonvisualization, is the rule. Rarely, the collecting systems will not be visualized due to vascular invasion. Calcification may occur, and renal carcinoma may mimic the lesion.

In contrast, no collecting system invasion is found in cysts. Characteristic infundibular elongation and calyceal cupping are the rule. Polycystic disease is rarely discovered in infancy. Leukemic infiltration is a much more common cause for bilateral renal enlargement in infancy and childhood than is polycystic disease.

Neuroblastomas, which arise in either the adrenal medulla or extra-adrenal chromaffin system, grow rapidly and may displace retroperitoneal as well as intraperitoneal structures. The intestines may be displaced anteriorly. The kidneys may be inferolaterally displaced. Calcification within the tumor and its metastases is relatively common. Bone, liver, and brain metastases are characteristic.

Retroperitoneal teratomas, especially in the sacrococcygeal or paravertebral gutters, are found in the very young. Adrenal tumors, such as the ganglioneuromas, et cetera, are rare. Adenomas and carcinomas may result in advanced skeletal maturation. One might be on the lookout for adrenal enlargement and/or renal displacement.

Lymphomatous and leukemic nodes may mimic pancreatic bed or other retroperitoneal tumors or cysts.

The characteristic posterolateral impression on the second portion of the duodenum in a jaundiced infant with a right upper quardrant mass is diagnostic for choledochal cysts.

Of the intra-abdominal intraperitoneal tumors, those of liver origin are extremely interesting.

Summary of Continuation Course presented September 27, 1956.

Embryonal hepatomas, cysts, and many rarer neoplasms are known to occur. The right lobe lesion, which is more common, is anterior and may displace the stomach to the left. The right kidney may be depressed. The hepatic flexure may be displaced downward and posteriorly. The left lobe tumor will displace the gastric fundus. Margulis described the undulated margin of the liver in hepatomas in contrast to the more sharply demarcated cysts. Rapid growth favors the diagnosis of sarcoma.

Mesenteric and omental cysts arise from the mesenteric root or mesocolon and are located anteriorly. Duplication cysts arise from the alimentary tract and usually do not communicate with the intestinal lumen. These often cause intestinal obstruction. Lymphangiomas of the mesentery may occasionally be confused with these.

Meckel's diverticula are rarely delineated radiographically. Abscesses, especially appendiceal, are often clearly demarcated as a distorted mass in the cecal bed. Rarely meconium ileus presents with a meconium cyst rather than with characteristic diffuse free fluid or calcification. Anterior pelvic teratomas arising from the genitourinary tract may have calcified parts or shadows from sebaceous material. The pelvic rhabdomyosarcomas and fibrosarcomas have less characteristic findings and present as retroperitoneal or pelvic masses.

Not all abdominal tumors or masses have been presented; nevertheless, from this selected material one can appreciate the role of the radiologic portion of the total exam in the establishment of a type specific diagnosis and localization of extra-alimentary abdominal lesions.

DETECTION AND TREATMENT OF NONINVASIVE CARCINOMA OF THE CERVIX

(Continued from Page 28)

conization or cautery may be considered with a view to preserving reproductive capacity; the same is true of observation alone. These two categories imply a degree of responsibility that many physicians do not care to assume, and no one can argue with their decision, which again is an individual matter.

Comment

It is apparent that the success or failure of this screening technique depends on many factors. However, the initial step, which is obtaining the smear, must be the responsibility of the general practitioner of medicine. No diagnostic clinic can reach all the population. No one else can successfully set up a long-term program for the community, for this cannot be a single examination but must be continued at intervals throughout the life of the individual woman. However, the process of obtaining and documenting smears is of no value if the chain of command does not follow through a well-trained cytotechnologist and an interested cytopathologist. Finally, the close cooperation of the pathologist and the general practitioner and free discussion between them are essential in the interpretation of some questionable smears.

It should be emphasized that the cytologic study of cervical smears is but one diagnostic measure; it is extremely valuable but it never is used alone to make the final diagnosis of carcinoma of the cervix. The gynecologic history, pelvic examination and careful inspection of the cervix cannot be discarded. Indeed, they assume even greater importance than previously.

The complexity of the interpretation of cytologic smears demands skilled personnel to read the slides. The errors inherent in poorly prepared material demand skill in obtaining the slides, and the human variables present demand the close co-operation of the pathologist and the patient's physician in deciding the proper treatment. The door is open on a new vista for women. Can we be reluctant to pass through?

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Clinical-Pathological Conference

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CASE PRESENTATION—MINNEAPOLIS VA HOSPITAL

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DONALD F. GLEASON, M.D.

A sixty-seven-year-old retired farmer and carpenter entered the hospital complaining of severe upper abdominal pain, which had begun suddenly at 10:00 p.m. of the previous night. For the previous six weeks, he had noted progressive weakness and a 20 pound weight loss, in association with a progressive difficulty in keeping food down.

Historical review disclosed that he had been troubled with intermittent bouts of severe heart burn and epigastric pain for over thirty years. This usually occurred shortly after meals. A peptic ulcer had been diagnosed many years ago and he had obtained occasional relief with the use of milk or antacids. The attacks of pain subsided spontaneously without therapy on many occasions also. In the early 1930's, three episodes of hematemesis and melena were experienced and similar bleeding recurred in February, 1952. Constipation had been a life-long problem, particularly during antacid ingestion and the patient was prone to use mineral oil in copious amounts. His maternal grandmother and seven of his mother's siblings had died of malignancies.

His first admission to this institution occurred in June, 1952. He had resumed his ulcer therapy after the episode of bleeding in February of that year, had noted progressive constipation and had returned to the use of mineral oil. In March of that year, he developed fever, chills and cough. His private physician was able to distinguish a left lower lobe infiltrate on his chest x-ray. This had not resolved after three months of antibiotic therapy and it was for this reason that the patient was admitted here. On bronchoscopy droplets of free fat were seen in the washings along with many inflammatory cells and lipid laden phagocytes. No malignant cells were seen on successive sputum specimens. There was no response to various antibiotics and a diagnosis of lipid pneumonia was made. A barium swallow and small bowel series were performed because of the patient's abdominal complaints and history of bleeding. A markedly contracted duodenal bulb and descending duodenum were noted. Beyond this point the duodenal loop and remaining small bowel were distended and contained air fluid levels. A barium enema revealed a normal colon and terminal ileum. Laparotomy was performed, but positive findings were limited to scarring of the duodenum and fibrosis of the mesentery of the terminal ileum. No resection was done. A diagnosis of diabetes mellitus was made on the basis of a postoperative glucose tolerance test, although no glycosuria was documented. He was placed on a convalescent ulcer regimen and discharged.

Three weeks later he experienced severe recurrent upper abdominal pains. These occurred approximately four times monthly thereafter and would often be relieved with an enema, although a hypodermic was sometimes required. The patient described the feeling as one in which his abdomen "seemed about to burst." The pain was non-radiating and was frequently associated with his periods of constipation. Hematemesis and melena had recurred thrice following his discharge here and had required hospitalization and subsequent transfusion once elsewhere. The third episode of bleeding following his first admission began with epigastric pain, bloating and vomiting. Four or five non-bloody emeses occurred during the night, to be followed by four hematemeses the next morning. The patient then returned here for his second admission in January, 1953.

At that time a 10-pound weight loss could be documented. The fasting blood sugar was 82 mgm. per cent. A qualitative test for urinary porphobilinogen was negative. X-ray examination of the chest, including planigraphy and bronchography, revealed no evidence of intrabronchial disease, but the diffuse process throughout the left lower lung showed little change. Repeat barium studies of the gastrointestinal tract also revealed little change. The cholecystogram, intravenous pyelogram, and electrocardiogram were all normal. Sputum smears and cultures and cultures of gastric aspirations were negative for histoplasma capsulatum and for acid-fast bacilli. He was treated conservatively and discharged. His bouts of abdominal pain continued and, although he was seen on subsequent followup examinations, little change in his status could be detected. His final admission occurred in October 1955.

The past history, system review and family history were believed to be otherwise noncontributory.

On physical examination the weight was 100 pounds, the temperature 99 degrees, the pulse 92 and the blood pressure 95/70. He was a small, emaciated white man with a high pitched voice and he appeared chronically ill. The right pupil was slightly larger than the left, as it had been on previous admissions, and reaction to light was sluggish. The right hemithorax was hyperresonant. Scattered sticky râles were described over the left lower chest. The vital capacity was 2.8 liters, slowly expelled. The testicles were small but the prostate was enlarged. A generalized hyporeflexia was described. The pedal pulses were not palpable. Proctoscopy was negative. The examination was felt to be otherwise within normal limits.

The laboratory examination revealed a white blood count of 8,700 with 68 per cent neutrophils, 26 per cent lymphocytes, 2 per cent monocytes, 2 per cent eosinophils and 2 per cent basophils. The hemoglobin was 11.5 grams per cent. The sedimentation rate

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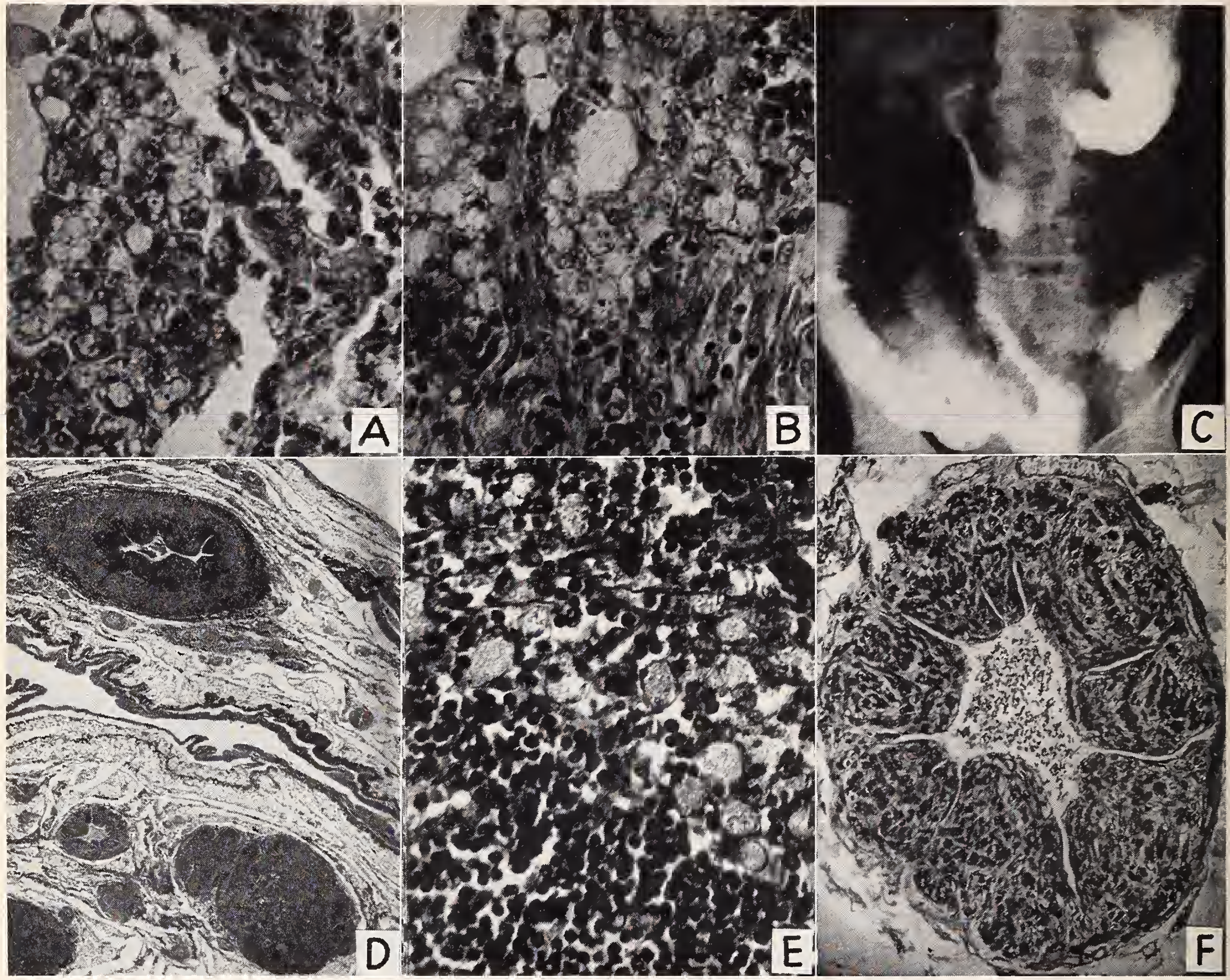

Fig. 1. Mineral Oil Excess

(a) Lipid pneumonia (b) Lipophages in spleen. (c) Barium study of upper gastrointestinal tract, revealing marked contraction of duodenal bulb and descending portion of duodenal loop. The remaining small bowel is alternately distended and contracted, and exhibits multiple air-fluid levels, with a bizarre mucosal abnormality.

(d) Section of mesentery near intestinal wall, showing proliferation of intima of small artery at top, with nearly obliterated lumen. (e) High power view of tiny lymph node at bottom of (d), showing lipophages. (f) High power view of tiny vessel between the two lymph nodes at bottom of (d), showing vacuoles within vessel wall and associated obliterative intimal proliferation. Photographs by Medical Illustration Service, VA Hospital, Minneapolis.

(Westergren method) was 67 mm. per hour. VDRL slide test was negative. Three urinalyses revealed a maximum concentration of 1.013. One plus glycosuria was noted on two specimens. Traces to 2-plus albuminuria were noted. Thirty to forty casts were noted in one specimen. The BUN was successively 12.5, 110 and 47.5 mgm. per cent. The fasting blood sugar was 99 mgm. per cent. The serum sodium was 145.6 and 130 meq/1. The serum potassium was 3.5 and 6.6 meq/1. The serum chlorides were 105.7 and 100.0 meq/1. The plasma CO_2 combining power was 23.0 meq/1. The serum albumin/globulin ratio was successively 3.9/2.3, 5.4/3.5 and 4.3/2.5 grams per cent. The serum alkaline phosphatase was 7 KAU. The serum acid phosphatase was 1.6 KAU with a prostatic fraction of 0.2 KAU. The serum calcium was 8.5 mgm. per cent and the serum phosphorus 2.5 mgm. per cent. The serum amylase was drawn during a bout of abdominal pain and was found to be 130 units. Two stools were negative for blood and meat fibers and the qualitative test for fat was negative. Twenty-four-hour urinary 17-ketosteroids were 3.6 mgms. Twenty-four-hour urinary 17-hydroxycorticoids were 6.6 mgms. Twenty-four-hour urinary gonadotropins were between 64 and 128 mouse units. The prothrombin activity was 57 per cent. The electrocardiogram now revealed a QS deflection in V2, 3 and 4, but was otherwise unchanged. X-ray examination of the chest revealed little change from previous admissions. Barium swallow now revealed a narrowed two inch segment in the distal esophagus and the remaining findings in the stomach and small intestine were still present. A barium enema revealed no intrinsic pathology of the colon or terminal ileum, but the sigmoid was displaced superiorly and to the right by a pelvic mass, possibly a distended urinary bladder. Catheterization yielded 800 cc. of residual urine.

Esophagoscopy was performed on the ninth day and a freely bleeding polypoid mass was seen just above the cardia. A biopsy revealed adenocarcinoma. The patient was transferred to the Surgical Service and an attempt was made to prepare him for resection. He continued to be unable to eat, however. He lost more weight, became progressively weaker and finally expired on the twenty-ninth hospital day.

Discussion

Dr. Frank M. MacDonald: I am going to assume that this audience has read the protocol and I will proceed with my analysis of it. This man was sixty-seven years old. He came into the hospital for the third time and died, after ten weeks of symptoms, of a carcinoma involving the esophagus. I prefer to discuss separately the disease involving the lung, the old peptic ulcer syndrome, the question of diabetes, his later course with abdominal pain, and the final carcinoma.

The first thing to be mentioned, I think, is the lung lesion. This is a left lower lobe infiltrate about which we have very little diagnostic information. It persisted apparently unchanged for at least three years, having been discovered early in 1952. There was very little change in spite of occasional treatment with antibiotics. The man was a mineral oil user and clinically this lung lesion was attributed to that. To go with that, oil droplets and lipophages were found in the bronchial washings. The value of this is limited by the absence of a statement that the man was prepared for such a study. Sputum studies and even bronchial washings are of less value in the diagnosis of lipid pneumonia if they are not preceded by a period of several days without the use of oily substances. There are certainly other possibilities for such a pulmonary infiltrate. Prior to the first films, the man could conceivably have had a pneumonia which had not yet resolved. He could have had an interstitial pneumonitis of the kind that we know rather little about. Since he died of a carcinoma in a nearby area, one might wonder whether a cancer of the esophagus had infiltrated the lung, but I am sure a three-year period is conclusively against this.

There are a couple of other things that appear in the history that I would like to dispose of early. One is the endocrine problem. The man may have had diabetes. His glucose tolerance test was done postoperatively and, therefore, may be unreliable, since a patient should be on an adequate carbohydrate intake for some time before the test. There are a couple of later sugar levels which don't help me much. So I am unable to make a definite diagnosis of diabetes mellitus. I must admit that it is a possibility. His 17-ketosteroids were somewhat low and he had rather high urinary gonadotropin levels. I am inclined to assume that this is, if not within the normal range, very close to it for a man of sixty-seven. The average daily urinary excretion of 17-ketosteroids in elderly males is approximately 4.5 mgms.[1] This indicates that this patient's value is not too far out of line. Am I right about that, Dr. Flink?

Dr. Flink: Yes, particularly for a person whose nutrition has been interfered with.

Dr. MacDonald: Some neurological findings are recorded. He had one pupil which was abnormal and did not respond to light, and hyporeflexia was noted. I think these findings, while not anything that one can diagnose, certaily suggest the possibility of some neurological cause for his abdominal pain and I will mention that later on.

He gave a history of peptic ulcer, having had, I assume from the protocol, recurrent abdominal pain for a period of about thirty years. The pain as recorded here was rather atypical for ulcer in that it came on shortly after meals and only occasional relief from ulcer medication and food was obtained. He had bleeding in the 1930's and in 1952, when first here, a duodenal deformity was noted. Subsequently a similar deformity was found. I am inclined to accept this as an ulcer history in spite of the atypical features. I do question whether or not it enters into the present problem—the problem of his abdominal complaints of the last three years. From 1952 on, it seems that something new was probably added. The first indication that we have was that he had the picture of air and fluid levels in the bowel. In June, 1952, he developed recurrent upper abdominal pain, accompanied by a sense of distention, which occurred apparently four times a month. He had at this time a negative abdominal exploration except for the old scarring of the duodenum. The way the pain is described in the protocol suggests that this is something new. I would like to know a little more about it if the information is available. First, apparently he had both bouts of pain and bouts of bleeding. I would like to know if they occurred at the same time or as separate incidents?

Dr. Johnsen: They did occur, on occasion, separately and not always simultaneously.

Dr. MacDonald: There was one in which the symptoms apparently occurred at the same time. Was the pain like that of intestinal colic? Do you know anything more about the duration?

Dr. Johnsen: Yes, the pain was colicky in nature.

Dr. MacDonald: Did he have nausea or vomiting during these attacks of pain?

Dr. Johnsen: Sometimes he did.

Dr. MacDonald: Do they describe objective distention of the abdomen during this time?

Dr. Johnsen: Yes.

Dr. MacDonald: Well, that helps me somewhat.

In 1955 there was a further change. The man was admitted having lost 20 pounds in weight. He was weak and unable to hold food down, and a diagnosis was made of an adenocarcinoma involving the lower end of the esophagus. In spite of attempts to prepare him for surgery, the man went on and died after a total duration of ten weeks from the onset of these symptoms, which I assume to date from the beginning of esophageal obstruction.

With the terminal episode there were renal findings including azotemia, variable amounts of albuminuria and some suggestion of limited specific gravity on three occasions. I can't make any specific diagnosis from this and I will have to assume that this is the sort of renal finding that may occur in seriously ill individuals for apparently a variety of reasons. There is an ECG change which is of some interest and I wonder if the cardiograms are available. I might also mention the displacement of the sigmoid by a pelvic mass which was thought to be possibly the bladder. The bladder did contain a large amount of residual urine so that I am going to assume that this explains the pelvic mass. The previous ECG was called normal and, after looking at this one, it seems to be as recorded in the protocol. He does have absent R waves across the precordium through V4 and no significant T wave or ST changes that I can see. May I see the x-rays now, please?

Dr. J. V. Testor (Resident in Roentgenology): There are a great number of x-rays on this patient. I have put up samples of his chest films taken from the three admissions. This is his original admission film in 1952 and we see the diffuse infiltrate in the left lower lung field described in the protocol. On the lateral view it seems to have involved the lower lobe and the lingula as well. Here is a picture of the chest in 1953 and I don't think there has been any essential change, except perhaps a slight increase in involvement. There are some increased markings in the right upper lobe now. This is a representative sample of a planigram taken in 1952 and it demonstrates that the main bronchial segments are open. There is no evidence of a hilar mass at that time. This is a film taken on the last admission and it shows essentially the same findings. We thought perhaps there might have been a slight progression of the infiltrate, but nothing very remarkable.

The most interesting x-ray feature in this patient is his gastrointestinal tract. This is a picture taken of the upper gastrointestinal tract in 1952 and it shows a marked duodenal deformity. The duodenum is markedly constricted and on all of our studies during the three-year period, it remains that way. So we are in complete accord with the diagnosis of duodenal ulcer. With this extreme amount of deformity it would be assumed that he had an active process. The most interesting feature, however, is his small bowel. This presents a strange picture. These films were all taken in 1952. First of all, there is a widening of the duodenal loop. The duodenum appears rather fixed in position and then as we follow the barium down into the ileum we get this very bizarre appearance, which is even present in the terminal ileum. One is rather hard put, here, to decide whether one is dealing with a process of mucosal ulceration or whether there is an obstruction or some process interfering with mobility, with resultant retained material within the bowel. We were inclined to choose the latter and we feel that it did not represent an ulcerated bowel but rather retained material within the bowel. This is an upright view of the abdomen taken in 1953. It shows multiple fluid levels and distention of the bowel.

Dr. MacDonald: Is gas seen in the large bowel?

Dr. Testor: Well yes, there is some in the large bowel but I think the fluid levels are principally in the small bowel. This picture doesn't look like regional enteritis and it doesn't have the appearance of conditions that we refer to as disordered motor function.

This is a barium enema taken in 1952 and it shows a normal colon. Another colon x-ray on his final admission is also normal except for the elevation of the sigmoid out of the pelvis. It could be on the basis of a distended bladder.

Here is the picture of the terminal esophagus and we have good evidence of an esophageal carcinoma. We have a segment of the esophagus which is narrowed and irregular with a sharp margination on its superior surfaces, which would go well with a carcinoma of the esophagus. The stomach looks the same as it did on the earlier films and the marked duodenal deformity persists.

Dr. MacDonald: Thank you. I am going to accept first of all the ulcer history, and I have no better pulmonary diagnosis than lipid pneumonitis. So my main problems are to relate his episodes of intestinal colic and the bleeding of the last three years, if I can, to the information at hand here. The bleeding was hematemesis and melena and therefore presumably from the upper gastrointestinal tract. I assume that the bleeding in the 1930's was due to his ulcer and that the protocol is accurate in indicating that there was a long lapse between that early bleeding and the bleeding that began to occur in the 1950's. The duration of the tumor through three years, if the tumor symptoms explain this recent period, seems rather long for a carcinoma of the esophagus. I would rather favor a carcinoma of the stomach which, as is well known, may begin in the cardia and only later invade the esophagus and cause obstruction. The x-rays do not show a mass in the stomach but this is notoriously a difficult area to examine. A harder thing to explain is the negative laporotomy, if he was already having bleeding from a carcinoma of the stomach, but again this particular area is a rather difficult one for an abdominal exploration and small carcinomas have been missed in this sort of situation.

A tumor of the small bowel also comes to mind with this picture. I think the one point most against this possibility is that the patient did not have the picture of recurring partial small bowel obstruction. He had gas in the large bowel on more than one occasion, but this amount of distention is supposed to be unusual for partial bowel obstruction, and the latter is not commonly thought to cause an appreciable filling of the bowel with gas. He could, however, have had recurring bouts of more or less complete obstruction which might conceivably have relented. The absence of a definite lesion in the small bowel on the x-ray does not rule one out since this is a rather difficult area for the roentgenologist to examine adequately. A point somewhat against a small bowel tumor also is that he had a primary lesion in the region of the esophagus or stomach, and this would give him a second primary tumor. Granting that these things do happen, we should nevertheless try to explain symptoms, if possible, with a single lesion. A hemangioma of the bowel might also be considered in a person that has had life-long bleeding and comes in with anemia. However, I have no other information from which to make this diagnosis. The possibility of an enteritis is actually more interesting to me now, after seeing the films, than it was on reading the protocol. The possibility of a widespread mucosal abnormality suggests this diagnosis. Regional enteritis does occur without the narrowing and formation of strictures and fistulas that we are all familiar with. I think that it is unlikely however. We have entirely too much small bowel and large bowel involvement here to be easily explained by this diagnosis. I would think it unlikely that enteritis could have been missed at the abdominal exploration. The possibility of a Meckel's diverticulum would have to be considered along with these other lesions of the small bowel, because it can bleed and cause recurring obstruction or intussusception, but I prefer not to make an extra diagnosis for which I have little support.

His pain was an obstructive type of abdominal or intestinal colic. This could be due to an organic obstruction such as the previously mentioned small bowel tumor. It could have been due to postoperative adhesions, although the first x-ray showing distention and fluid levels was made before his abdominal exploration. The possibility of an inhibitory or paralytic ileus ought to be mentioned, but one would not expect this much pain and vomiting and, since he definitely had colic, this can be ruled out. The possibility of a neurological lesion is of interest here since the man may have had diabetes and also had hyporeflexia and pupillary changes. Either a tabetic or diabetic pseudotabetic neuropathy might be a conceivable background for this sort of picture. The constellation representing diabetic neuropathy seems to have three major components. Diabetic nocturnal diarrhea is one of these and does not seem to be in the picture here. Secondly, these people may have periods of constipation with some degree of abdominal pain. I haven't seen any mention in the literature of anything approaching this degree of colic. Gastric dilatation and occasionally dilatation further down in the bowel represent a third manifestation of diabetic neuropathy but again this does not seem to represent the picture we have here. As far as tabes is concerned, I am afraid the scanty neurological findings, along with the negative VDRL, while not conclusively against this diagnosis, certainly do not support it well.

The last possibility might be a spastic ileus, which is a rather rare condition characterized by localized spasms with hyperactive bowel above the region of spasm. This resembles very much the picture of organic obstruction. Many people say that these conditions cannot be differentiated clinically before abdominal exploration. There is a special type of spastic ileus that I was interested to learn about in preparing for this discussion. It can occur with carcinoma in the region of the upper abdomen, particularly carcinoma of the pancreas, although apparently other lesions have been reported. This has been called O'Gilvie's syndrome by Dr. O. H. Wagensteen in the new edition of his book "Intestinal Obstructions."[2] What appears to happen is that the patient may have extensive carcinoma in a fairly solid area of the pancreas or nearby structures. When the malignancy invades the region of the celiac axis and picks up some of the autonomic nerve supply to the bowel, a picture of recurring bowel obstruction of the sort one gets with a spastic ileus occurs. I am afraid that I can't make this diagnosis either, much as I would like to, because the syndrome went on for a three-year period and it seems unlikely that a person would have this much carcinomatous infiltration and survive for that period of time. None of the cases reported have survived for more than a period of months.

Dr. MacDonald's Diagnoses

1. Adenocarcinoma of the gastric cardia invading and obstructing the esophagus
2. Duodenal ulcer
3. Lipid Pneumonia
4. Spastic Ileus

Dr. E. B. Flink: There is one thing that I have learned about these protocols. They usually don't give you a case unless they have an anatomical lesion to demonstrate. This isn't always true. They may fool you sometimes and throw in one with a functional explanation but they usually have a pretty good anatomical reason for the patient's symptoms. I think the possibility of an internal abdominal hernia causing intermittent bowel obstruction has to be considered in addition to the other anatomic possibilities discussed. I wonder if there is any information about his diet. Was he on a carefully managed ulcer diet all the time?

Dr. Johnsen: Only when troubled with symptoms.

Dr. Flink: What sort of dietary habits did he have? Do you have any information about that?

Dr. Johnsen: No, except that he consistently lost weight during the years he was cared for here.

Dr. Flink: One of these films looks like it might show a Meckle's diverticulum. Is that possible?

Dr. Testor: I suppose it is possible but it may just be an overlapping of the intestinal loops. We felt that there was some pathology involving the small bowel. It looked similar to a peritoneal carcinomatosis but it didn't seem very likely that a carcinoma would show up three years later than the bowel symptoms. Therefore we considered amyloidosis or some collagen disease involving the bowel.

Dr. Flink: I wish to compliment Dr. MacDonald on not mentioning those possibilities. I just wonder if the possibility of a bezoar in the lower ileum could be considered. That is why I asked about the dietary habits. He did have what looked like retained material on one of his films. A bezoar, however, is usually in the stomach and I have never heard of one in the ileum.

Pathologic Findings

Dr. Gleason: I think we have a case here of something that is frequently talked about but is never demonstrated.

At autopsy there was no jaundice, edema, ascites, hydrothorax or pericardial fluid. The heart was small, weighing only 180 grams. This happens frequently in chronic diseases. The heart very seldom weighs under 250 grams unless there is some very serious chronic illness. The coronary arteries were widely patent and there were no myocardial scars.

The left lung weighed 650 grams. Multiple round, yellow-white areas were noted in it, particularly in its lower lobe. In contrast to the left lung, the right lung weighed 350 grams. Its lower lobe contained a few small calcified nodules and a smaller number of the large pale areas present in the left lung.

The gastrointestinal tract was of primary interest here. In the lower end of the esophagus was an obvious tumor, which extended upward about two inches as a flat plaque. It also extended downward a short distance into the cardia of the stomach. Several of the lymph nodes in the immediate area were a little larger than normal but were not obviously malignant and no tumor was found anywhere else in the body. The small intestine was dilated very markedly, from the ligament of Treitz to the ileocecal valve, and the wall of the small intestine was quite tough and leathery in many areas. In many other irregular areas it was quite discolored, to the point of complete blackening and obvious gangrene. The abdomen was distended by this dilatation of the small bowel. It smelled very bad. The striking finding was that the branches of the mesenteric artery were all very prominent, being of the pipe-stem type, with very narrow lumina. When cut, the arterial branches stood out like little pencils. We dissected the mesenteric arterial tree quite thoroughly and could find no thrombosis. The whole process grossly seemed to be a very severe, diffuse atherosclerosis. Post-mortem culture of the peritoneal fluid, of which there was only a very tiny amount, was a tribute to the care with which this culture was taken. It remained sterile for five days.

The liver was very small, weighing only 850 grams. The spleen weighed 90 grams, another instance of atrophy in chronic disease. The gall bladder, pancreas and adrenals were normal. The kidneys were quite small, weighing about 50 grams each. They showed a little coarse scarring. The testes were both very small, being about 1.5 to 2.0 cm. in diameter.

Medical Resident: Were both superior and inferior mesenteric arteries involved with the atherosclerotic process?

Dr. Gleason: No, just the superior mesenteric artery. On microscopic examination, lipid-containing macrophages are seen in the sediment from a bronchial washing. The presence of lipid may be confirmed with fat stains on the fresh material. A section of the lung reveals some emphysema, but most of it shows a severe lipid pneumonia, being almost solidified by masses of lipid-containing macrophages in scar tissue.

The same phagocytosis of lipid is seen in the spleen. We don't usually have the history of excessive mineral oil ingestion so well documented. All of the lymph nodes from the abdomen contained macrophages with ingested lipid, presumably mineral oil. The liver also contains cells filled with lipid. They aren't quite as convincing there, because we normally see a few fatty cells, but these don't have the usual central distribution. They are just scattered around haphazardly. They are not particularly portal or central in location.

A section of the stomach shows a rather thin carcinoma extending along the mucosa a short distance into the wall. It is an adenocarcinoma and it is presumably from the stomach.

In the testes the seminiferous tubules are all completely atrophic. The Leydig cells are preserved. This is the picture we anticipated from the hormone levels.

Dr. E. T. Bell: Do you want to say something about the hormones here, Dr. Gleason?

Dr. Gleason: Well, I think it has been covered. The gonadotropin level is increased and the 17-ketosteroids are diminished because the testes are atrophic. He also has a fairly large prostate.

A section of the peripheral mesenteric arteries reveals their thick walls, some of which are connected by scar tissue. The tiny collapsed lumen is much smaller than normal for vessels of this size. This is because of marked collagenous thickening of the intima. No actual occlusion of the lumen by this intimal proliferation or by thrombus could be demonstrated. There wasn't any striking amount of arteriosclerosis elsewhere in the body.

Medical Resident: If you could have confirmed the presence of diabetes mellitus, such a severe degree of atherosclerosis could be better explained.

Dr. Gleason: The arteriosclerosis was limited to the mesenteric artery. This is the main reason we presented the case. We feel that this is an instance of chronic arterial insufficiency of the small intestine due to arteriosclerosis. One often hears this cited as the cause of some of the abdominal pain that accompanies coronary artery disease, but very seldom, if ever, is it demonstrated.

Medical Resident: What do the small intestines look like microscopically?

Dr. Gleason: Early changes of infarction are seen in the sections from the worst areas. No scarring or other change is seen.

Medical Resident: Is there a lot of fat in the mucosa?

Dr. Gleason: No, there is not. Whipple's disease can be excluded.

Medical Resident: What did the origin of the superior mesenteric artery look like?

Dr. Gleason: There weren't any big plaques at its origin. The sclerosis was a diffuse process in almost all of the branches of the superior mesenteric artery and extended out to the bowel. A review of sixty cases of occlusion of the mesenteric artery from various causes appeared in the literature in 1954. Fifty-one of these were definitely due either to thrombosis or embolus. Three of them were due to periarteritis. Two were due to septic arteritis. The remaining four had severe arteriosclerosis of the mesenteric arteries. Two of these had small thrombi which were not related to congestive heart failure. The remaining two were caused by an incomplete closure, like this case, and had gangrene of the bowel, but they were both instances of congestive heart failure. We often see severe sclerosis of the mesenteric arteries with no clinical signs or symptoms, but we have never seen this degree of sclerosis before. We feel that this is a case of chronic ischemia of the small intestine, due to diffuse arteriosclerosis of the superior mesenteric artery.

Medical Resident: What explanation do we have for the bleeding?

Dr. Gleason: It could have come from the carcinoma in the gastric cardia. The duodenum showed a scarring deformity and we couldn't even find the spot where the ulcer had been. We don't have any other explanation for the bleeding unless it resulted from the arterial insufficiency causing localized areas of the small intestine to become infarcted.

Final Anatomic Diagnoses

1. Adenocarcinoma of the gastric cardia, with invasion of the esophagus
2. Duodenal scarring
3. Lipid pneumonia
4. Cachexia with atrophy of vital organs
5. Localized atherosclerosis of the peripheral branches of the superior mesenteric artery
6. Infarction of the small intestine

Dr. Johnsen: In completing the discussion of this case I would like to mention the possibility that the chronic and copious ingestion of mineral oil may be of more importance in the pathogenesis of this man's symptoms and findings than we realize. For many years it was believed that mineral oil passed through the alimentary tract without being absorbed. Yet lipophage aggregates have been noted in this patient's liver, spleen and mesenteric lymph nodes. In recent years such findings have been reported in other patients who have used mineral oil regularly. If we acknowledge, then, that mineral oil is capable of leaving the intestinal lumen, either by direct infiltration or by lymphatic or hematogenous transport, we must at the same time find it curious that the superior mesenteric arterial bed of this elderly man was the only major vascular system seriously involved by atherosclerosis. Furthermore the site of greatest involvement appears to be peripheral in location, including even the smaller vessels near the intestine, and sparing the more proximal major vascular trunks. It is possible that the lumina of even the smallest vessels in the mesentery have been compromised by intimal proliferation in response to mineral oil deposition within the vessel wall. If so, the resultant ischemia of the intestinal wall, especially in response to an increased demand for blood after ingestion of food, could easily explain this man's postprandial colic. It could also explain the unusual intestinal spasm and mucosal irregularity that puzzled the roentgenologists.

Finally, I would like to propose the possibility that this patient's cachectic state (which was documented even before the presence of his carcinoma) and the small size of his vital glands and organs may have been related to faulty assimilation of valuable nutrients, because of an excessive mineral oil ingestion. We are now reviewing our experience with the complications of mineral oil usage and expect to report a series of such cases in the near future.

References

1. Freeman, H., et al: Steroid Replacement in Aged Men. J. Clin. Endocrin. & Metab., 16:779-789, 1956.
2. Wangensteen, O. H.: Intestinal Obstructions. Springfield: Charles C Thomas, 1955.

PHYSICIANS TAKING GRADUATE WORK

More than 30,000 physicians took graduate work either as interns or residents during the 1956-57 academic year, according to the AMA Council on Medical Education and Hospitals.

The figure is another increase from previous years and indicates the results of increased medical education facilities throughout the nation.

The report also showed an increase of 31 per cent in the number of internships offered, and a raise in average monthly stipends paid to interns. In hospitals affiliated with teaching institutions, the raise was from $87 in 1954 to $140 in 1956. The average stipend for non-affiliated hospitals increased from $136 to $177 over the same period.

Public Health

REVISED BIRTH AND FETAL DEATH CERTIFICATES

A. B. ROSENFIELD, M.D., M.P.H., Minneapolis
J. W. BROWER, LL.B., M.A., Saint Paul

A statewide maternal mortality study has been carried out by the Maternal Mortality Committee of the Minnesota State Medical Association, beginning first in 1941. All deaths due directly to obstetric causes, all deaths associated with but not directly due to pregnancy, as well as all deaths occurring within ninety days postpartum (whether obstetrical or non-obstetrical) have been intensively studied. Results of the study have been presented to staff and medical society meetings and have been utilized in undergraduate and postgraduate education. These data have been of value to the medical profession in the continuing reduction of maternal mortality.

Thus, while there is accurate statewide data on obstetric mortality there is none on morbidity, except for limited studies in individual hospitals. For example, how early in pregnancy do women obtain prenatal care? What are the complications of pregnancy and their frequency? How many pregnant women are Rh tested? There is no available information as to the incidence of Cesarean sections, preeclampsia, hemorrhage, or Rh complications in Minnesota. We know the facts in maternal deaths as a result of the maternal mortality study. But, we have no statewide data on maternal morbidity. There are only gross estimates based on very limited and localized reviews of case histories. There is thus a need for brief, easily available and obtainable obstetrical data.

To make it possible to obtain current data regarding the complications of pregnancy, labor and delivery a detachable confidential section has been added to the bottom of the certificate of fetal death, beginning January 1, 1958 (Fig. 1). A similar confidential section will be added to the birth certificate at a later date. With few exceptions only check marks are necessary to answer the questions.

There are four main columns: (1) pregnancy, (2) not related to pregnancy, (3) labor, and (4) operative procedures. At least one item should be checked in each column. either "none" or the appropriate condition. For example, in column one, under conditions related to pregnancy either "none" should be checked, or if there were complications, the appropriate condition should be checked, such as preeclampsia, eclampsia, hypertensive disease, nephritis, pernicious vomiting, pyelitis, anemia, hydramnios, or multiparity. If a complication not listed was present during the pregnancy the item "other" should be checked and the particular condition should be written in. Each of the four columns should be checked in a similar fashion, noting either "none" or the appropriate condition.

The first day of the last normal menses as well as the date of the first prenatal visit are requested so that information will be available to determine how soon women seek prenatal care. Information on Rh testing and the results are essential to determine the extent of the Rh problem in Minnesota. The three last questions pertain to the frequency of birth injuries, congenital malformations (noting the specific type) and the results of the blood serology.

The information requested can be secured, quite reasonably, by the hospital staff, it can be checked very quickly, and it is most relevant to current problems of maternal health. The addition of the confidential section has been approved by the Maternal Health Committee and has received the approval of the Council of the Minnesota State Medical Association.

The completion of this confidential section will make it possible to tabulate and analyze data on the births and fetal deaths in Minnesota. The analyses of data involving some 80,000 births and 1,800 fetal deaths annually should provide valuable information on the frequency of normal and abnormal pregnancies, the frequency and variety of obstetric complications, as well as those associated with pregnancy and labor. The confidential section should be detached from the certificate and mailed directly to the Section of Maternal and Child Health, Minnesota Department of Health. It will be used only for research purposes. Physicians are protected in such studies under the Minnesota confidential research law.

To obtain complete and accurate information requires the thorough study of clinical records by a trained staff. Even a limited study of an adequate sample of the more than 80,000 live births would require considerable time, staff, and funds. This is the preferred approach but is not feasible at this time. Until such time as such a study becomes possible, these questions on birth and fetal death certificates will throw some light on the many problems in maternal care.

Dr. Rosenfield is Director of Division of Special Services and Mr. Brower is Deputy Executive Officer, Minnesota Department of Health.

CONFIDENTIAL MEDICAL REPORT

ATTENDING PHYSICIAN: After completion, detach this section and MAIL DIRECTLY to the Section of Maternal and Child Health, Minnesota Department of Health, University Campus, Minneapolis 14, Minnesota

This information is to be used only for scientific purposes of the State Department of Health

Complications of Pregnancy, Labor and Delivery (Check at least one item in each column)

Related to Pregnancy

- ☐ NONE
- ☐ PREECLAMPSIA
- ☐ ECLAMPSIA
- ☐ HYPERTENSIVE DISEASE
- ☐ PERNICIOUS VOMITING
- ☐ ANEMIA
- ☐ HYDRAMNIOS
- ☐ MULTIPARITY
- ☐ OTHER — SPECIFY
- ☐ HEMORRHAGE
  - ☐ ANTEPARTUM
  - ☐ INTRA PARTUM
  - ☐ POSTPARTUM

Not Related to Pregnancy

- ☐ NONE
- ☐ HEART DISEASE
- ☐ DIABETES
- ☐ SYPHILIS
- ☐ TUBERCULOSIS
- ☐ PYELITIS
- ☐ HYPERTENSIVE DISEASE
- ☐ NEPHRITIS
- ☐ OTHER — SPECIFY

Labor

- ☐ NONE
- ☐ PLACENTA PREVIA
- ☐ PREMATURE SEPARATION OF PLACENTA
- ☐ PROLAPSE OF CORD
- ☐ ANOMALY OF CORD
- ☐ BREECH PRESENTATION
- ☐ OTHER MALPRESENTATIONS — SPECIFY
- ☐ CONTRACTED PELVIS
- ☐ OTHER DYSTOCIA
- ☐ OTHER — SPECIFY

Operative Procedures

- ☐ NONE
- ☐ OUTLET FORCEPS
- ☐ LOW FORCEPS
- ☐ MID FORCEPS
- ☐ HIGH FORCEPS
- ☐ CESAREAN SECTION
- ☐ BREECH EXTRACTION
  - ☐ PIPER FORCEPS
- ☐ INTERNAL VERSION AND EXTRACTION
- ☐ OTHER — SPECIFY

Date of first day of last normal menses

Date of first prenatal visit

Was mother blood tested for RH factor? NO ☐ YES: Positive ☐ Negative ☐

Mother previously RH sensitized? YES ☐ NO ☐

Previous RH neg. baby NO ☐ YES ☐ Living ☐ Dead ☐

Other blood dyscrasias: Specify____________________

Serologic Test for Syphilis: YES ☐ Result__________________ NO ☐

Birth Injury to Infant: NO ☐ YES ☐ If yes, describe:

Congenital Malformation of Infant: NO ☐ YES ☐ If yes, describe:

It is recognized that physicians are busy, and therefore, they will not be queried regarding unreported items in the confidential report. However, it is recognized that physicians are vitally interested in improving the standards of care and so it is anticipated that they will cooperate voluntarily in this simplified report, knowing that the data will be utilized for study. Periodic reports of the findings will be disseminated to physicians for their information and guidance. We know of no other method which, for the same amount of effort, can secure so much pertinent data. The accurate completion of this confidential section will be most useful in studies of obstetric and newborn experiences, and will contribute to medical knowledge.

UNIVERSITY HOSPITALS REPORT BUSIEST YEAR IN HISTORY

University of Minnesota Hospitals cared for more patients during the year ending June 30, 1957, than in any previous year, according to the annual report of Ray M. Amberg, hospitals director, made at the annual staff meeting, December 20.

A total of 14,953 bed patients were admitted during the year ending June 30, 1957, and 119,683 visits were made to the out-patient department.

In the 1956-57 period, 925 more patients were admitted than during the previous year, and out-patient visits increased 3,314 visits over 1955-56. Average stay of bed patients was 12 days, in comparison to the 13-day average stay a year ago.

Daily average population of the hospitals during the last fiscal year, Amberg reported, was 508—six fewer patients per day than for the year before. Days of hospital care provided patients during the year were 185,434, as compared with 188,284 in 1955-56.

The University Hospitals' total of 666 beds—78 of them in the Variety Club Heart Hospital—will be increased by 80 beds next fall, when the Masonic Memorial hospital will be completed.

The cost of operating the hospitals' in-patient department still is rising, Amberg pointed out in his summary. Expenses per patient day during the year ending June 30, were $29.83, an increase of $1.65 over the previous 12-month period.

Surgeons performed 9,922 operations in University Hospitals during the year, an increase of 1,137 over the previous year's total of 8,785 operations.

The 119,683 visits to the out-patient department include those of 20,377 new patients. During the 1955-56 fiscal year, 116,369 out-patient visits were recorded. Cost per patient visit last year amounted to $5.98, an increase of 20 cents over 1955-56.

In operation since 1909, the University Hospitals serve as a training ground for more than 500 medical students in addition to more than 900 student medical technologists, nurses, x-ray technologists, physical and occupational therapists, social workers, dietitians, pharmacists and dentists. Their work is carried on in the hospitals and out-patient department under the direct supervision and guidance of a staff of full-time and part-time instructors.

University Hospitals rules require that each patient must be referred for admission by his family physician or by a physician in his local community.

Editorials

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

GOVERNMENT

Last month I pointed out that government, like medicine, is dedicated to the welfare and happiness of human beings. Both are essential to our well-being. Lack of adequate medical care can result in pain, suffering, and death. Lack of adequate and effective government can result in poverty, unrest, crime, and even war. Unless governments today can work out programs and policies that can prevent nuclear warfare, our present civilization and future generations are threatened. Truly, in government today, we need the utmost in ability, integrity, and understanding of human problems.

Yet, as I have pointed out, whereas the practice of medicine engages only those who are highly qualified and well trained, the practice of government involves every voter as well as those elected to public office. Citizen participation in government is of increasing importance. I believe that we can make a valuable contribution to better government by encouraging and increasing such citizen participation on many different levels.

The primary level of citizen participation in government occurs, of course, on election day. But I would urge, not only more informed voting, but also increased citizen participation in the partisan political activity that precedes election day. For politics is an integral part of government, and our party system is absolutely essential to the functioning of our free, democratic government.

Another level of citizen participation which, I believe, offers great possibilities is that of co-ordinated community action to solve community problems. We have, for example, begun to develop such participation to solve problems of children and youth. Last year in Minnesota, town meetings on these problems were held in 162 communities. The participants made recommendations which were later discussed in our state-wide Governor's Conference on Children and Youth.

Such citizen participation involves not only state and local governmental agencies, but also volunteer organizations and individuals that are interested. People participate on a level at which they are familiar with problems and needs. Their volunteer services are of immeasurable value, and are willingly and eagerly given.

Perhaps one of the most effective areas in which volunteer services have assisted in Minnesota, in a function for which the state government holds responsibility, is in the field of mental health. In co-operation with every mental hospital in the state, citizen volunteers have made invaluable contributions by their services to patients. Their activities have brought about a far better community understanding of the problems and needs in the mental health field.

I am hoping that we can develop co-ordinated volunteer citizen activity on a community level to meet and solve many of the problems of our increasing numbers of older citizens. Such co-ordinated programs should include activity in the fields of education and recreation, health, employment, housing, and many others. Medical advance has increased our life span phenomenally in the last few years. It is our responsibility to see that the added years are happy and productive ones.

Finally, I have found that citizen participation on special state-wide committees can make invaluable contributions to government. The Governor's Advisory Committee on Vocational Rehabilitation illustrates such contributions. Outstanding physicians on this committee have given of their time and ability to develop a greatly improved program for the rehabilitation of the physically handicapped in Minnesota.

All these forms of citizen participation are of real value to government in that they help to provide services essential to our people. And because they bring about a better understanding of government and a deeper interest in its problems, they make more effective citizens of those who participate.

Orville L. Freeman,
Governor

A RATIONALE FOR PROTEIN

The emphasis of today's advertising on protein as a nutrient has increased the nutritional guidance the physician must give to his patient.

One reason for the popularity of the term *protein* in advertising campaigns may be the concept held by laymen that protein lacks calories and is, therefore, particularly suitable in the reducing diet. This concept has become so well established, one is seldom reminded that each gram of protein furnishes four calories: the same amount of energy that is furnished by carbohydrate. The isocaloric character of these two nutrients should be a basic part of any nutrition story.

Today's advertising also dwells on the specific dynamic action of protein. Even physicians sometimes fail to remember that this stimulatory effect, which can be demonstrated in experimental animals fed a pure protein diet, is nullified when protein is combined with fat and carbohydrate as it is in any human diet.

Protein consumed over and beyond that required for growth and replenishment of tissues is deaminized. This leaves a non-nitrogenous residue which, like carbohydrate, may be used for energy. An excess beyond energy needs can be stored as body fat. Since protein is the most expensive part of any diet, its use to fulfill the roles of carbohydrate and/or fat is most wasteful. Current research is focusing on more economical ways to achieve an ideal balance of nutrients in which carbohydrate and fat are present in sufficient amounts to save protein for its specific functions of building and repair.

Under consideration at present are the amino acid contributions of the "partially incomplete" proteins in plants when these are supplemented with small quantities of the "complete" proteins usually found in animal foods. Excellent evidence indicates that humans need less of the so-called "complete" proteins in conjunction with the "partially incomplete" proteins than experimental animals do. Rats, for example, which were fed a predominantly bread diet (found adequate for a group of European children) failed to grow as well as the control animals receiving a diet higher in protein.

This finding was part of a study by Drs. R. A. McCance and E. M. Widdowson* who attempted to determine the relative nutritional values of whole wheat and white flours of various extraction rates. Approximately 300 children (ages four to fifteen) were maintained for a year on a diet consisting mainly of bread made from flour, water, yeast, and salt only. These investigators observed excellent growth and no signs of nutritional deficiencies during the year's duration of the experiment, even in the groups which received 75 per cent of their protein from white bread. They found that for these children, the average intake of 9 grams of animal protein and 6.5 grams of protein from plant sources, other than bread, adequately supplemented the bread protein.

After twelve months of observation the children were divided into two groups. One group served as controls while the other received a milk supplement which almost tripled their animal protein intake. This phase of the experiment lasted for six months. The control group of children, receiving no milk supplement, developed to the same extent as those receiving the supplement—further evidence of the protein adequacy of the predominantly bread diet.

The protein of enriched bread made in the United States approaches that of the predominantly bread diets of McCance and Widdowson. Enriched bread, on the average, is made from a formula requiring 4 pounds of nonfat dry milk for each hundred pounds of flour. Investigations under way reveal that this ratio of milk protein to wheat protein provides a combination of amino acids which, on the basis of present knowledge, approaches the nutritional ideal.

American Institute of Baking

THE SUPREME COURT'S BLOW TO McCARTHYISM

The nine men who compose the Supreme Court of the United States come slowly and cautiously to their decisions. The case of the Californian Communist leaders who were acquitted last week began as far back as 1952. And the Court has just handed down rulings on other cases which also date back to the years when what is commonly called McCarthyism was sweeping the country.

What causes such delay? There are three reasons. First, a case moves ponderously through the complex machine of American justice—appeals, motions for re-hearing, new trials and review. Secondly, the Court itself is overworked. As a matter of fact, the justices are now putting in overtime to clear their calendar. Lastly, as one justice remarked to me in a recent conversation, the Court does not rush its fences. Knowing that a great decision may have great consequences, may indeed reshape American society as well as American law, the justices will not lightly revoke laws passed by Congress or interfere with the actions of the government. Sometimes they will find grounds for a judgment which evade an important constitutional issue, or a straight challenge to existing law and practice.

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*McCance, R. A., Widdowson, E. M.: Studies on the Nutritive Value of Bread and on the Effect of Variations in the Extraction Rate of Flour on the Growth of Undernourished Children. Medical Research Council. Report No. 287. London, England: 1954.

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Yet, when they are put to the direct question, the justices can act with courage and imagination—virtues more striking, perhaps, because some of them have little or no judical experience on which they can fall back to justify the positions they take. They may be men like Chief Justice Warren, who have had long careers in politics, or university teachers such as Justice Frankfurter, or men whose lives had been spent in public service. Whatever their personal background or experience, however, they each bring something to their office—and each gains something from that office whether a man has been a Republican or a Democrat, whether he is a Catholic, Protestant, or Jew. Southerner or Northerner, the office elevates and dignifies him.

The Constitution drafted nearly 200 years ago has continually to be adapted and interpreted to the needs of modern Americans and that is done by the new men of the Supreme Court. That is why, in their current judgments, as in the sweeping decision three years ago that pronounced the death sentence upon the segregation of Negro school children, the justices must weigh social and political considerations along with the legal precedents.

In the past two or three weeks they have weighed the causes and the consequences of McCarthyism, and found that the extreme measures tolerated when anti-communism was at its peak in the United States do not match up to the standards of toleration and civil liberty required by the American Constitution. No one should minimize the impact of these rulings. In effect, the Court has destroyed the Smith Act, under which most prosecutions of leading and lesser Communists were launched. It has told the Government that it is not enough to charge Communists with advocating a conspiracy to overthrow the Government of the United States—there must be proof of a real conspiracy: merely to show past or present membership of the Communist Party will no longer constitute grounds for conviction. The Court has also made it much more difficult for the Federal Bureau of Investigation and prosecuting attorneys to rely on hearsay evidence and on informers; it has ruled that a man cannot be forced to answer every irrelevant or contentious question that comes into the heads of congressional investigators; and it has insisted that academic freedom implies the right to explore even opinions that are politically unpopular.

It is too soon to predict how these decisions will work out in detail. I lived in the United States when what is loosely called the "witch hunt" was in full cry. Some things were done then—by the Government, by congressmen, by state legislatures and even the lower courts—which seemed to threaten American liberties. The Court has now declared in scorching terms that such abuses were reprehensible, avoidable, and unconstitutional. Is it late in the day to come to that conclusion? Not at all. It was clear to me when I was in the United States recently that the heresy-hunt was failing fast, and that public opinion was reacting sharply against its excesses. More recently still, the trial of the playwright Arthur Miller has evoked much critical comment. The Supreme Court has now given point and direction to that new feeling.—Norman Mac Kenzie, "At Home and Abroad," *Home Service.*

CAN YOU BE INSURANCE POOR?

The Established Physician

The physician who has practiced from five to ten years and who has been at least moderately successful, usually will have some specific objectives for his financial and estate plans, by now and will have taken some steps to carry out those plans. Certainly he will consider what further steps should be taken to keep pace with his objectives, to keep his plans in proper balance and to keep them workable.

By this stage of his business life he will have an account or two at the bank, a few bonds and some life insurance as fixed value investments. He may own his home, an automobile, office equipment and perhaps a share in a partnership, as variable investments. It will be to his benefit to review his plans periodically to avoid leaning too far in one direction or the other.

The amount of life insurance the physician now owns will have been determined by the operation of many factors, including his responsibilities, earnings and personal beliefs in life insurance as desirable property. If in his early years of practice he has family responsibilities and a desire to have an estate for the protection of his wife and youngsters (in case he is taken away from them prematurely) he will recognize the logic of buying his estate on the installment plan, just as he is buying a home and car on the installment plan. Life insurance provides him with the opportunity to do just that—but without the risk of payments falling due after death or during disability, as is the case when other property is purchased on the installment plan. If he cannot buy the entire estate he wants even on the installment plan (and he needs $30,000 to $40,000 more than his neighbor who has Social Security to do the same job), he may purchase part of it with permanent life insurance and "rent" the balance with term insurance. When he "rents" part of the needed estate he will be given an option to buy that portion on a permanent basis at a later date when income permits.

A physician in his early thirties after having practiced for three or four years, told the life underwriter that he needs an estate adequate to provide the following if he were to have a fatal accident or be taken from his family prematurely by some other cause:

Estate clearance fund .... (including a $15,000 home mortgage)	$20,000
Annual net income for 20 years...........	4,000
Education Funds for his 3 sons....	5,000 for each

He was told an estate of approximately $100,000 was needed to do the job. He was unable to buy an estate of that size even on the installment plan because of the annual installment required. However, he could afford $75.00 per month for family security. With this premium he could purchase $25,000 permanent life insurance and "rent" $75,000 with term insurance. And, by renting the $75,000 he preserves the right to make the $75,000 or any portion of it a permanent part of his estate, later, when he will be in a better position to pay the installments required by a larger permanent estate.

Is he "insurance poor," as some of his friends say, because he owns $100,000? Or, isn't he veritably "insurance rich" without paying premiums that would make him "poor?"

Even at that he may be "leaving his change on the counter" if he does not take advantage of the settlement options on his insurance and the accompanying tax advantages in arranging the paycut to his family following his death. The entire proceeds, of course, could be paid out in one sum to the widow. But is an arrangement a good one when it places on the widow the responsibility of deciding how to use this large sum of money at such a critical time?

Consider this $100,000 insurance estate and the job it is intended to do. It is anticipated that $20,000 will be needed to settle the estate including the mortgage on their home. The balance of $80,000 paid out over the growing-up years and providing $5,000 for education of each of three sons can be set up in several ways. Under one arrangement this $80,000 will pay out in excess of $100,000, including over $20,000 of taxfree interest, taking advantage of a new provision in the Internal Revenue Code of 1954. Under another arrangement the same pay-out of $100,000 from $80,000 of proceeds will put the $20,000 interest in a taxable category. However, both arrangements have the advantage of experienced management by the life insurance company and both provide an assured income.

Lack of proper planning has been costly for many insurance buyers, causing them to "leave their change on the counter" while creating hardship and misunderstanding for their beneficiaries.

Whether or not you want to have your life insurance planned in this way, is up to you. But it costs no more to use the settlement options and the allied services of the insurance company to carry out your wishes for the financial security of your family.

Herbert F. Mischke, C.L.U.

SAFETY GLASS AND YOUR CAR

When considering safety factors afforded by the glass in your car, it is well to assure yourself that you have the finest quality of glass, that you have the utmost quantity of vision available, and that you keep your windows clean and in good operating conditions as is the universal rule with all other safety equipment.

But there is a further important factor in your driving safety—your eyes. You need the kind of glass and the controlled conditions which will afford you the most comfort and the least fatigue.

Investigations at the Mayo Clinic have led to the conclusion that 25 per cent of an individual's total physical energy is used in the simple task of seeing. Poor visual conditions increase that percentage. Difficult seeing tasks call for more energy. A driver threading his way through heavy traffic at 40 to 50 mph certainly calls for more physical energy for seeing than does a person sitting in a chaise lounge in a serenely shaded garden area looking at flowers, trees and guests.

Therefore, the first helpful hint to the motorist is to be sure that he has safety plate glass in all his car windows. The brain automatically corrects for visual distortions so that most of the time we are not conscious of the continual outpouring of physical energy just to see reasonably clearly in our moving car. It stands to reason that the clarity of vision through safety plate glass saves much of energy wasted in looking through wavy or slightly distorted window glass.

Glare from the bright sky is another cause of fatigue on the highway. The modern automobile offers methods of reducing this cause of driver inefficiency. First, there are adjustable sun visors to shade the eyes. But they have the occasional temporary disadvantage of hiding a suspended traffic light unless viewed at a considerable distance.

So, through the windshield glass, another device is offered to reduce sky glare. Beginning at a point above the driver's line of vision for viewing traffic conditions, the broad windshield has been shaded with a band of neutral color, blending well with the natural tones of the sky. This shading is an almost imperceptible tone adjacent to the clear area of the windshield and deepens toward the top of the windshield. The light meter reading is almost halved by this method. Also this neutral color enhances color values and makes it even easier to differentiate between the red, yellow and green colors in traffic lights.

This shaded windshield comes in combination with a special type of heat-absorbing plate glass which has the added physical property of filtering out the infrared rays which carry a large part of the heat energy of the sun. Because of its slightly greenish tint the question has sometimes been raised as to its effect on driver vision. Many important and exhaustive tests have been conducted over more than ten years on this particular point.

Most thorough of these tests indicated that, at night, visibility was reduced *at the threshold of vision* by 3 per cent. Thus if a driver, looking through a standard windshield, were able to see an object in the light of his headlights at a distance of 200 feet, the same driver, looking through a heat-absorbing windshield would be able to see the object at 194 feet. If in both cases, he was traveling at 40 mph, and if in both cases his eyes were focused on the exact spot where the object first became visible, he would see the object 1/10th of a second later, looking through heat absorbing glass. But if, as would almost always be the case, his eyes were focused elsewhere on the road until the object was within the threshold of vision—say at 190 feet, he would see the object through either windshield at exactly the same time. From that point on, as the object is approached, there is no measurable difference.

Since the tinted and shaded glass tends to reduce the fatigue from sky glare during daytime driving, the driver who goes on into nighttime driving is less eye-weary. Most scientists feel that his relative freshness would more than compensate for the almost negligible measurable difference, leaving a balance of safety favoring the heat-absorbing windshield. There are between twelve and one-half and fourteen million cars now on the road with the tinted safety glass purchased as an extra option. A large percentage of car owners who prefer the glare-reducing tinted glass are repeat purchasers basing their preference upon personal experience.

Thus by his choice of the safety plate glass in his car one may add glare reduction, eye comfort and reduce normal driving fatigue.

WILFRID HIBBERT
Libbey-Owens-Ford Glass Company

CONFESSIONS OF A HOBBYIST

Music

It is, perhaps, belittling to write of serious music as a hobby, because it becomes a part of one's life. My love of music goes back to boyhood in Cambridge, Massachusetts, where, as a matter of routine, everybody went to hear the Boston Symphony Orchestra and the Kneisel Quartet. I studied piano eight years, and organ three, and, when I went to Harvard, took every course in heoretical music offered—harmony, counterpoint, orchestration, free composition, and the rest. I knew I'd never be good enough to make music a life profession, but comprehending something of how music is put together has added to my enjoyment a hundredfold.

In the first school where I taught, they had a small orchestra. Also, they had a big double bass, and nobody to play it, so I was drafted. A seventy-five-cent book "How to Play the Double Bass" was my instructor, and I sawed away on the big fiddle for five years. I doubt if I could play a note on it now, but I still work, intermittently, at the piano, with fingers still limber from years of typing my own letters. Mrs. Briggs is a real musician, a fine performer, and that creates great pleasure for me.

I marvel at the number of people who seem to enjoy listening to music with the naive idea that a composer does no more than create a succession of pleasant sounds. As I look at a great audience in Northrop Auditorium, I sometimes wonder how many of those there know that the first movement of almost every classical symphony is constructed as formally as a sonnet, or a good fifteen-minute speech. There is an introduction, a main subject, a bridge passage and a secondary subject, and this section is usually repeated so that the listener may know the material. A second section develops that material, and third section, almost identical with the first section, ends the movement with a peroration, called a "coda." If you know this, your whole picture of the music is clarified. Other movements have other conventional forms, not at all hard to recognize.

I have shown to groups of men and boys through the medium of projected photographs of orchestral scores (home-made) and phonograph records, how even a person who knows nothing of music can learn quickly to follow score. Up is up and down is down, and the structure becomes clear to the ear through the eye. I think I was one of the first to use this method, now widely used.

John deQ. Briggs

By providing a program of postgraduate education and a fuller utilization of educational media, medical societies, state and local, can do much to stimulate continued improvement in the quality and the thoroughness with which cancer detection practices are carried out in their respective areas.

JOURNEY INTO SPACE

Stockholm was on the brink of a major Trade Fair and a European Heart Conference, so of course taxis were at a premium. Our train for Lapland left at 4 p.m. and we felt quite sure that it would go without us, but by dint of vast patience, extravagant arm-waving and a large slice of luck, a taxi with an illuminated LEDIG sign drew in to the kerb, and we had met and conquered our first obstacle. The station was a seething mass of people—residents, tourists, trade delegates, and an extraordinary number of physicians from the most unlikely countries. We tracked down our platform at last, and found a motley crowd of cardiologists surrounded by mounds of luggage, and a small neat woman guide in the uniform of the Swedish tourist agency which had organized this pre-conference trip to the Arctic Circle. With enviable efficiency and an amazing command of most of the European languages, she shepherded her protégés toward their train, and in the minimum of time we found ourselves in a minute, but beautifully equipped sleeper.

The journey started without undue incident, and the hours of early evening slipped away while we watched, entranced, the Swedish countryside, lit by the sunset, unfolding beyond the dusty windows of the train. We had spent an intriguing half-hour exploring our compartment which the Swedes, with their passion for mechanical contrivances, seemed to have fitted with an incredible quantity of buttons, levers, and other more complicated gadgets, and we had discovered, to my chagrin, that the exciting-looking door which appeared to lead at the very least to a private bathroom, was thoroughly locked. This was borne out when the occupants of the next sleeper in their turn tried to force it open, and we found that it was in fact nothing more than a communicating door between the two. We enjoyed, a little later, an excellent dinner in the restaurant car—a meal which was enlivened by the appearance on the menu of reindeer steak, and by the delight with which the small Swedish girl opposite us achieved, in a burst of unsuspected erudition, an English "Thank you." After dinner we settled down in our compartment with our staple holiday reading, a couple of thrillers, but life was

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not to be without its own small excitements. At intervals the door into the corridor opened abruptly and heads poked themselves in to announce the sale of magazines, or tobacco, or, for all we knew to the contrary, merely to bid us good-night. This habit of walking in unannounced, though pleasantly companionable, had its hazards when the time came to get ready for bed, and it was a considerable relief to find that there was actually a lock we could use. G. turned it, and for a while all was peace. I had just got myself into a nightgown, not entirely Victorian in character, and was sitting on the edge of the bunk, brushing my hair, when an ominous clicking sounded from the corridor and I found myself facing the unembarrassed stare of a large man in official uniform. He said something politely but quite uselessly, and disappeared whence he came, leaving G. and me in a state of mingled consternation and giggles.

We were due at Riksgränsen around tea-time the following afternoon. The tour programme had anounced grandly that we would be staying at the Lapplandia Tourist Hotel, from which we would make excursions, and the prospect sounded inviting, though perhaps a little incongruous in association with the wild, mountainous country through which we were now passing, with its lakes and peaks, and almost entire lack of civilization. I think, with the traveller's unquenchable optimism, we imagined suddenly rounding a bend and coming upon a charming little Swedish town, situated in beautiful unspoilt country, but oh most certainly boasting every comfort that the twentieth century can offer. The actuality was of course that we found ourselves deposited at a tiny wayside station, with untrammelled nature spreading in every direction, the only apologetic gesture that humanity had made being the railway line, a ski-lift, and a group of wooden houses that resolved themselves, upon closer inspection, into the Lapplandia Tourist Hotel—and as we left the train, it started to snow! In actual fact the Lapplandia was a ski-lodge, and an extremely comfortable one, with central heating, excellent food, and the most delightful staff that one could imagine, and having said good-bye to our visions of a smaller edition of the Grand in Stockholm, we did not regret them once—but we were a little disconcerted by the surrounding countryside. The lovely view from our windows of the deep, placid lake, the snow-clad peaks, and the unspoilt stretch of richly glowing tundra was well enough, and indeed we praised it to the skies from the asethetic point of view, but exploring it was another matter. Not one of the party, some forty strong, seemed to have conceived of the possibility of there not being such a simple amenity as a road, and it came as something of a shock to find that the much-vaunted excursions were not of the motor-coach, or even of the boat, variety, but on our own feet. The women, particularly, had no footwear suitable for trudging through snow-covered heath, and worse. But a little matter of that kind was of small consequence. The Lapplandia staff came to the rescue in no uncertain manner and by the first morning, every cardiologist and cardiologist's wife, was equipped with something that would do at a pinch—and in some cases, did just that! I was lucky enough to get stout walking shoes that fitted perfectly, but G. had to be satisfied with zip-up galoshes, which he first tried wearing over bedroom slippers, but finally padded with two pairs of thick woolen socks.

Chief of our excursions was the great Lap hunt. On our first walk, a nonchalant stroll over a sort of goat-track lasting a mere couple of hours, we were accompanied by a Swedish guide and a bounding Alsatian—presumably to do a St. Bernard act if anyone fell by the wayside. The guide stopped every quarter-mile or so and gazed at the surrounding peaks like stout Cortez in Darien, but as he spoke nothing but Swedish, and the members of his party could raise between them only English, French, German, Spanish, Portuguese and Egyptian, it was some time before we discovered that he was looking for Laps to show us. It is a curious fact that, like flowers in an English garden when one visits it, one never seems to go to a place at the right time to see its chief attraction, unless of course it happens to be something immovable, like St. Paul's, and so it was with us and the Laps. Almost all we knew about Lapland before we visited it was that it was famous for the Midnight Sun, reindeer and Laps. We were, inevitably, out of season for the Midnight Sun. The reindeer which apparently had appeared in scores to those at the front of the train, had all taken fright and disappeared by the time our rear coach arrived on the spot—and now there was not a Lap in sight. Our poor conscientious Swedish courier, who had promised us Laps, like lollipops to children on a school treat, was deeply concerned, and in a fashion that seemed to us nothing short of miraculous, finally discovered that A Lap Family was still in the vicinity. The method she used involved complicated telephone conversations, but as telephoning a Lap encampment seemed about as fantastic as ringing up a Hottentot village, we forbore to go into the matter further. Enough that the Laps were there, and that we were to walk out and see them, reindeer skin tent, traditional costume, and all. The distance, we were told, was a mere four kilometres following a short train ride, and having hurriedly sat down and computed what it meant in mileage, we decided it was worth it.

We set out, accordingly, all forty of us, arrayed in a variety of garments that for piquancy and colour would have put any Lap to shame—it was cold enough to need plenty of clothing, but heavy overcoats would have been a disadvantage when walking, and the problem was solved with duffle coats, windbreakers, mackintoshes, and every kind of woolly. I worked out my own salvation by wearing G's pyjama coat under my sweater. The odd couple of feet which had to be turned up at sleeves and waist practically gave me another layer. In due course, we arrived at the end of the rail journey and, marshalled by our two guides, who were heavily laden with luncheon packs, we set out across the tundra, like a pale imitation of the Great Trek. The first stop came quickly and abruptly. We drew up in a group inside a large stockade, and were informed impressively that this was where the reindeer were corralled and branded. Naturally at another time of the year! We gazed round obediently for what seemed like about twenty minutes, but without discovering any new fact about the stockade, and then away we went again. This time there was no stopping. The path, if one might dignify it by such a name led away from the railway line, across marshland, over rocky outcrops, down greasy slopes, over perilously balanced duckboards which as likely as not, acted as a seesaw and plunged one into muddy swamp. Here and there a red mark on a boulder suggested the way, but for the most part one trusted in Providence and the guides. The views all the way were magnificent, provided there was time to look at them, and one was not busy with one's feet. After the first few miles the party had divided into three—an energetic vanguard, who had obviously spent all their lives trekking across the wide open spaces in their various homelands, an American couple and ourselves in the middle, acting as a link, and a large and weary group coming on behind in a sort of pilgrim's progress, groping and stumbling and picking themselves up again. The hills stretched away interminably ahead, and the path to the Laps apparently did too. Breakfast was a long way behind, and lunch seemed destined to be far in the future. Now and then we had a moment of bafflment when the party in front seemed to have been swallowed up by the ground, but patient tracking of footprints would bring us up with them again, and finally, rounding a shoulder of rock, we came upon them all on the shore of a river. This had to be crossed in two leaky boats which could only carry about five at a time, and it was a hazardous process, to say the least. One of the boats had literally to be emptied after each crossing, and both needed constant baling while in mid-stream. However, to my permanent surprise, we all got over safely, and embarked upon the next part of our travels. Equally surprisingly, for we had now got used to the idea of walking for ever, this was a comparatively short stretch, and there at last was the Lap tent. Not exactly a thing of beauty, perhaps, but one cannot have everything, and it was picturesque in its way, with the flap open, a fire burning brightly inside, and a forest of little birch trees surrounding it cozily. The male Lap, the only one in evidence when we arrived, came entirely up to expectation with his brilliant costume of the traditional red and blue, and it was exciting to drink coffee made from his cheerfully hissing kettle, and to eat our long overdue lunch in the atmosphere of nomadic domesticity which surrounded the tent. Some washing hung on a roughly-made arrangement of birch logs, and we had not long to wait before the "housewife" appeared with the rest of the family. There were "oohs" and "aahs" of admiration for her gaudy embroidered dress, and the two children were surrounded in an instant. They too wore the red and blue garments that characterize the national costume, the baby boy's cap surmounted by an enormous scarlet pom-pom which made him look like some fantastic doll. Cameras clicked madly, coins changed hands, and the weary pilgrims relaxed comfortably in the knowledge that, at whatever cost, they had at last seen the Laps. It was a pity that a few of the more enterprising spirits, looking for fresh fields to conquer, walked to the top of the slope overlooking the tent, and found on the other side, The House that the Laps Really Lived In, though disillusionment would inevitably have followed when something was produced from the depths of the tent and carefully handed round, and turned out, to our everlasting mortification and delight, to be a Visitors Book.

After this, the walk home could not fail to be a little of an anti-climax, particularly perhaps for a gallant Egyptian who suffered from arthritis and had spurred himself to the effort with an extra dose of cortisone. However, as someone remarked, it made an excellent exercise tolerance test for those cardiologists who believe in practicing what they preach.

Patricia M. Bourne

The author is the wife of Dr. Geoffrey Bourne, chief cardiologist at St. Bartholomew's Hospital, London, England.

A LIVING TRUST

Let's say, simply, that it is a trust you set up during your lifetime, rather than one you might provide for in your Will. You sign a trust agreement—and your bank signs it; you turn over the securities or other properties to the bank to be held in trust—and the trust is "in business." Some people call it an *immediate trust,* and that is perhaps a better title. But "Living Trust" is standard.

Of course, your question is part of a larger question: What is a trust? Any kind of trust, whether founded on a Will or trust agreement?

The *legal* title to property is transferred to a trustee. However, the trustee has no *beneficial* interest in the property—only duties. His duties are to manage the property—prudently; to make it produce income—the highest income consistent with prudence; and to pay that income to some designated person or persons. (These are known as "income beneficiaries.") The document creating the trust—reading somewhat as your Will might read—will direct that upon the happening of certain events, such as the death of the income beneficiaries or their reaching stipulated ages, the trust shall be terminated. Then the trustee shall distribute the property, in such proportions as the trust document may direct, to a designated person or persons. (These are called "remaindermen.")

The same person may be both income beneficiary and remainderman, as in the case of a son for whom a trust is created: the income to be paid to him until he is, say, thirty years of age, at which time—if he is living, of course—the principal is to be paid over to him. And the same person can be both the maker of a trust and its income beneficiary. Thus *you* can set up a Living Trust for your own benefit as long as you live—or until you decide to change or revoke it.

At this point you ask:

How can a Living Trust benefit me?

An immediate and measurable advantage is *management.* A trustee has a double job. A fair rate of income must be earned. At the same time the principal must be conserved. For the protection of income beneficiaries and remaindermen the law imposes certain standards of care upon trustees. They cannot deal with the property as if it were their own; they must remember always that it really belongs to you or your beneficiaries

Therefore, banks make a *business* of trusteeship where experienced people guide the selection of trust investments. Markets and economic trends are studied; specific industries and particular companies within such industries are analyzed. The studies are continuous. Then decisions are made—not by one man but by a responsible committee Nor are decisions isolated; they are made within a framework of investment policy determined by a committee designated by the bank's Board of Directors.

You get *group judgment,* therefore—not individual whims, prejudices, or hunches. And back of the judgments are research and analysis.

Now, of this management you can utilize as little or as much as you please. You can reserve all ultimate decisions to yourself—or you can give the trustee sole responsibility; and there are all sorts of in-betweens.

Perhaps management is all you will require leaving you free to devote *undistracted thought* and *uninterrupted time* to the demands of your business or profession. But a Living Trust can do far more than this for you.

After your death it can carry on just like a Will The income can be paid to your wife—to your children—or others; and eventually the principal will be disposed of as the trust agreement may direct. And of course it is not necessary that you yourself be a current income beneficiary. Many Living Trusts are made for the immediate benefit of others; wives, children, grandchildren, relatives

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Nothing in the above is intended as legal advice.

urther removed—anyone to whom an obligation felt, or whom there is a desire to aid.

Thus a Living Trust may be set up to benefit charity"—a word broad enough to cover any reliious, educational, scientific, philanthropic or ther purpose which is for the good of the public t large. Such a trust is often called a "Foundaion"—but it is just a variety of Living Trust. ometimes individuals are the immediate benefiiaries; the "remainder" goes to charity. There is much more that we can tell you about charitable oundations if you are interested.

You may ask:

Can I save taxes with a Living Trust?

You can—under certain circumstances. And lmost any kind of Living Trust, whether or not t saves taxes, can save many expenses usually nvolved in the settlement of estates.

First, it should be explained that there are two inds of Living Trusts. One is *revocable:* you can hange or end it whenever you wish. Such a trust will save *you* no taxes; even though the income s paid to someone else, it will be treated as your ncome for tax purposes, and upon your death, the rust fund will be taxed as if it were part of your state. But such a trust may pass free of estate axes from the next income beneficiary to the ultimate recipients of the principal; and as mentioned above, certain expenses of estate administration may be eliminated or reduced. Not the least of the advantages of the Living Trust as a *Living Will* is that there is no interruption of the management of assets, or of the collection and payment of income, as there is in the settlement of estates by Will. Without recourse to any court, and normally without publicity, the Living Trust carries on for the benefit of those next in line.

The other kind of trust is *irrevocable.* You can't get the trusteed property back and you can't change the income beneficiaries or remaindermen or shift their interests around.

An *irrevocable* trust is ordinarily made for the benefit of others of your family, and the designation of such beneficiaries is usually permanent and unchangeable. Such irrevocable gifts in trust may result in reduction of income tax burdens and savings of estate taxes, since present gift tax rates are lower than estate tax rates. However, careful thought should be given before making irrevocable gifts in trust. As a rule a revocable trust is preferable because it can be changed to meet changing conditions.

Your final question may be:

How do I go about setting up a Living Trust?

As a first step, your bank can help you with the basic planning involved in the practical and financial aspects of living trusts. This can be most useful to you and to the lawyer of your own choice who will draw the trust agreement. He will tell you what you can do under the law, and how you should do it, to gain the maximum advantages taxwise—and otherwise. (There is much to trusts besides taxes.)

And of course—it goes without saying that your bank would be very pleased to serve as your trustee.

THE AMERICAN NATIONAL BANK
OF SAINT PAUL

MECHANISM OF THE ERYTHROPOIETIC EFFECT OF COBALT

It has long been known that cobalt ion increases the rate of erythropoiesis and eventually produces and maintains a polycythemia. More recent work has demonstrated that the earlier suggestion that cobalt exerted its effect by making the bone marrow anoxic is untenable. With increasing attention being paid to the role of the plasma factor, erythropoietin, in the control of erythropoiesis, and with the availability of simple, short assays for this hormone, the authors have investigated the possibility that cobalt may be effective through erythropoietin production. The data presented indicate strongly that cobalt enhances red-cell production by increasing formation of erythropoietin.

Male rats were given subcutaneous injections of $Co^{60}Cl_2$. Ten hours later, the animals were bled and enough blood withdrawn so that 20 ml of "cobalt plasma" was available for assay. The amount of cobalt remaining in the plasma was 0.21 micromole/ml. The cobalt plasma was assayed by the Fe^{59} incorporation method in starved rats using normal plasma with the same amount of $CoCl_2$ as the control. The incorporation of Fe^{59} into the red blood cells of rats that had been treated with these preparations of plasma was compared with that of rats treated with plasma from animals made anemic by phenylhydrazine.

The pronounced increase in the incorporation of Fe^{59} which was produced by the cobalt plasma cannot be the result of the presence of a small amount of cobaltous ion but can be interpreted as being the result of an increased amount of erythropoietin in the plasma. When erythropoietin has been characterized more fully, it will be possible to determine whether cobalt plasma contains the factor identical with that found in anemia plasma.

The authors accumulated evidence showing that certain of the properties of erythropoietin in anemic plasma are also common to those of the active factor in cobalt plasma.

While these parellelisms are not conclusive evidence that the material in cobalt plasma is identical with that in anemic plasma, they suggest that both types of plasma contain erythropoietic factors with grossly similar properties.—Goldwasser, E.; Jacobson, L. O.; Fried, W., and Plzak, L.: *Science,* 125:1085 (May 31) 1957.

Horatio B. Sweetser, M.D.,
President, Minnesota State Medical Association

President's Letter

A LOOK FORWARD

At the beginning of this new year, your new president brings greetings to the members of the Minnesota State Medical Association. He had hoped that this year might be passed in a relaxed fashion and that greetings could be pleasant and happy and that the routine work of the Association would go along without incident.

However, every year brings its problems and crises, and this year is no exception. In fact, your new president is a little awed by the responsibilities which will be placed on all of us. The goal of "what is best for the patient" always, with respect to the medical profession in this country, has been best approached by the private practice of medicine within the limits laid down by law. This method has seemed best to the profession, not only for the patient but for the community and for the state. A few years ago, those who thought that compulsory health insurance and government medicine gave better promise were defeated in their endeavors. Now government medicine is again advocated, this time for a smaller part of the population and with appealing features.

In the closing days of the last session of Congress, Aime Forand of Rhode Island introduced a bill, HR-9467, which would provide Federal Government payment for hospital and nursing home services and for surgery not of an elective nature. This payment would come from taxes paid by almost everyone to the Old Age and Survivors Insurance Trust Fund; i.e., Social Security funds, so called. This, of course, is a compulsory tax. Payments would be made for the care of anyone receiving, or eligible to receive, Social Security benefits. Everyone over sixty-five years of age, most women over sixty-two years, and the totally disabled over fifty years are eligible. Hospital care would be paid up to sixty days a year, or nursing home care up to 120 days a year, and surgical fees would be paid provided that the surgery is done by surgeons certified by the American Board of Surgery or by members of the American College of Physicians. Eligibility is based only on attainment of a prescribed age, regardless of income, and, therefore, invited reduction in the prescribed age. This, therefore, can be an introduction to compulsory hospital and surgical insurance for everyone.

There is a certain emotional appeal in the provision for added care for the aged. There is no question that provision must be made for this group, which now numbers 12 to 13 million. There should still be some responsibility carried by children, though the feeling of this responsibility is apparently becoming less. There are other ways of caring for the aged than by compulsory insurance.

Some, perhaps many, of the president's letters will be devoted this year to the work of the various committees of the State Medical Association. The threat of government medicine inherent in the Forand Bill cannot be met only by opposition. Medicine must cooperate and should be in the forefront in the effort to find other, more reasonable, more American ways of caring for the older citizens. Our scientific committee on "Geriatrics and Chronic Illness," with Dr. Leo A. Nash of St. Paul as chairman, is intelligent and active, or the Committee on Medical Service has here a golden opportunity to lead in the development of ways to care for the aged. County society committees on aging can cooperate with all the other activities in this field. Medicine, while of great importance, is only one facet in this problem, so effort must be made to join with all the others in the field.

If we all join together, we can accomplish two great things: defeat another attempt to socialize medicine, and, at the same time, take care of the old people in our midst who need care the most.

Horatio B Sweetser

President, Minnesta State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

FEDERAL HEALTH SPENDING

During the current fiscal year, the United States will spend approximately two and one-half billion dollars for all health and medical purposes. This is about the same amount as last year.

No less than twenty-three United States cabinet departments and independent agencies are engaged in some medical operations and approximately seventy-nine separate health-medical activities worthy of listing and describing. The Department of Health, Education and Welfare tops the list of all departments in health-medical spending with $849,394,800. The Veterans Administration is spending $849,374,000 within $20,000 of HEW, but Defense Department this year drops back more than $80 million to $702,000,000, largely because the decreasing size of the armed forces means fewer uniformed men and dependents to care for.

Next comes Atomic Energy Commission, but its medical spending of $40 million—mostly for research—is far down the column from the Big Three.

International Cooperation Administration—$37 million. Nineteen additional departments and agencies have substantially less, the last item—the $12,145 allocated to the congressional physician.

AMA MAPS ALL-OUT CONGRESS FIGHT TO PROTECT MEDICINE'S INDEPENDENCE

Effective opposition to the pending Forand Bill is possible only when members of the medical profession are well-informed.

In the interest of preservation of free enterprise in the medical profession, the medical press throughout the nation is conducting extensive informative campaigns in order that the pending legislation may be more effectively opposed.

An effective presentation is given in the following article by Dr. J. Lafe Ludwig, member of an AMA committee on legislation. Your earnest consideration is encouraged.

Confronted with new Congressional threats to the private practice of medicine, the American Medical Association is girding for an all-out fight to preserve the profession's independence under our free enterprise system. At issue are proposals introduced in the last session to liberalize the Social Security Act to permit hopitalization of the aged, with the costs paid out of Social Security funds.

AMA Takes Action

The AMA Board of Trustees and the AMA Committee on Legislation, realizing the dangers inherent in such proposed legislation, have taken steps to combat the bills in the Congressional session convening in January. Acting on a recommendation of the Legislation Committee, the Board's Executive Committee approved appointment of a task force to give special attention to the issue. Edwin S. Hamilton, M.D., board chairman, appointed George M. Fister, M.D., of Ogden, Utah, as chairman of the task force and named the following members, all of whom have accepted: Frank C. Coleman, M.D., Des Moines; Robert L. Novy, M.D., Detroit; George F. Gsell, M.D., Witchita, Kansas, and James Duffy Hancock, M.D., Louisville. The new committee, with assistance of the various AMA departments, will conduct research designed to gather information on the problem of hospitalization of persons over sixty-five years of age. The task force will not be called upon to prepare or present testimony or engage in legislative activities in opposing the pending legislation. These functions will be performed by the committee of which I am privileged to be a member.

Free Government Medical Care

Focal point of the forthcoming fight is HR-9467, introduced by Rep. Aime J. Forand, Democrat of Rhode Island. This bill would increase the benefits payable under the Federal old-age survivors, and disability insurance program and provide insurance against the costs of hospital, nursing home and surgical service for persons eligible for old-age and survivors insurance benefits. The proposed law provides that a person eligible for surgical services could freely select the surgeon of his choice provided that the surgeon must be certified by the American Board of Surgery or must be a member of the American College of Surgeons. . . .

Forand Receives AFL-CIO Endorsement

Congressman Forand, addressing the House on August 30, explained that he had introduced the bill in the waning hours of the last session to permit my colleagues in the Congress and individuals and groups to give the proposals thorough study in the period before the opening of the next session of Congress in January. Forand added: I am gratified that President George Meany of the AFL-CIO has endorsed my proposed amendments as necessary, sound and enlightened.

Need For Bill—Forand

Forand claimed there is evidence of the need for higher cash benefits and for better methods of meeting medical costs of the aged. . . . He said that early in the next session he intends to present an analysis of such evidence based on the current situation and the latest available studies. It is this so-called evidence that the AMA task force will attempt to refute by data collected in its survey. The proposed amendments relating to health benefits for OASI beneficiaries deserve special attention, Forand stated. They have been limited deliberately to facilitate enactment of this much-needed measure in 1958.

Increased Benefits Proposed

Forand's bill would increase cash monthly benefits about 10 per cent and the principle of relating benefits to earnings would be reapplied by increasing the earnings ceiling for contribution and benefit purposes from $4,200 to $6,000 a year. For a person with average monthly earnings of $200, therefore, the new primary insurance would be $85.80 instead of $78.50. With a $6,000 wage base ceiling, the maximum individual benefit would become $151.80 instead of $108.50. Discussing his proposed new program of health benefits, Rep. Forand said: They (the eligible aged) would be entitled to sixty days of hospital care and to subsequent skilled nursing home care, up to a total of 120 days in a twelve-month period. The cost of their surgical care would also be covered, with free choice of qualified doctors. According to best available estimates, this bill would add about 1 per cent of payrolls to present level premium costs. Contribution rates for employer and employee would therefore be increased by .50 per cent each in 1959, and the self-employed would pay .75 per cent more on their incomes up to $6000. The increase now scheduled for 1960 would be postponed to 1961.

Free Drugs, Appliances, Medical Care

Forand said his program would provide hospitalization and nursing home care for many who are not able to pay for private insurance against the cost of such care. Twelve or thirteen million persons would thus receive the protection of such payment in the first year, Forand told the House. Whether or not they retired, men would be protected at age sixty-five, if they had made sufficient contributions in covered employment or self-employment. Women would similarly be eligible at age sixty-two without having to take the actuarial reduction in benefits provided in present law. Forand said that hospital services, which would be paid include the services, drugs, appliances and medical care ordinarily furnished by the hospital to its bed patients. He added:

Free Hospital Care

The insurance would cover semi-private accommodations if available unless other accommodations are required for medical reasons—in short, those services ordinarily provided in plans such as the Blue Cross. Such services would be covered when provided by a licensed hospital which had entered into an agreement with the Secretary (of Health, Education and Welfare), except that the costs would not be paid for care in: any tuberculosis or mental hospital, any Federal hospital, or any other hospital for hospital services which it is obligated by contract with the United States to furnish at the expense of the United States; or any hospital furnishing services at public expense, except when a person receiving such services must meet a means test. . . . Services provided by a nursing home would be covered by the insurance provided by the bill only if the individual has been transferred to the nursing home from the hospital on a doctor's certification that the services are necessary for an illness or condition connected with that for which the hospital was treating him.

Free Surgical Care

Surgical services which would be paid for include those provided in a hospital and which are certified as necessary by a licensed physician. Oral surgery would be included when provided in a hospital and certified as necessary by a licensed physician or dentist. Surgical services provided in the outpatient department of a hospital or in a doctor's office would be included in case of emergency or for minor surgery. Fees would be established by agreement between the Secretary of Health, Education and Welfare and qualified doctors, either individually or through medical associations, Forand said. Nothing in the provisions of the bill or in agreements with hospitals, nursing homes or physicians shall be construed to give the Secretary of Health, Education and Welfare supervision or control over, first, the practice of medicine or the manner in which medical services are provided; second, the details of administration or operation of hospitals or nursing homes; or third, the selection, tenure or compensation of hospital or nursing home personnel.

FORTY-FIVE MILLION UNDER FORTY STILL HAVE NOT HAD SALK VACCINE

According to recent statistics released by the National Health Survey, 45,000,000 persons under forty years of age still have not received Salk poliomyelitis vaccine. The new data, based on information obtained in household interviews, also shows the following: 5,000,000 under forty received one injection, in contrast to earlier estimates of 17,000,000; 25,000,000 have received two shots, instead of the earlier estimate of 28,000,000; the number receiving the full three shots has increased from an estimated 28,000,000 to 34,000,000.

HIGLEY RESIGNS AS VA ADMINISTRATOR

Harvey V. Higley has resigned as administrator of Veterans Administration, asking that he be relieved of his duties "as early as convenient." Mr. Higley, who has headed the VA since July 22, 1953, wants to return to private life. In accepting the resignation, President Eisenhower said, "you have performed a valuable service . . . outstanding achievements have been brought about under your capable administration." Mr. Higley was chairman of the board of the Ansul Chemical Co. of Marinette, Wisconsin, before coming to VA.

ESSAY CONTEST PARTICIPATION URGED

The Minnesota State Medical Association will participate in the local sponsorship of the 1958 Association of American Physicians and Surgeons' Essay Contest for high school students.

The state association is offering $200.00 in prizes for the four best essays in Minnesota and the Association of American Physicians and Surgeons offers $2,675.00 in fourteen national prizes. The first prize is $1,000.00.

"The Advantages of Private Medical Care" or "The Advantages of the American Free Enterprise System" are essay topic choices.

Essays must be completed by March 1, 1958, in order to be judged at a county level before March 15, 1958, and at the state level, before April 1, 1958.

Dr. J. A. Cosgriff, Sr., Olivia, has been named the chairman of the state committee. He has written the officers of every society urging them to encourage the society to sponsor the essay contest in all local schools. The Woman's Auxiliary has agreed to do the leg work and help sponsor the project.

Already Kandiyohi-Swift-Meeker Medical Society is offering $25.00, $20.00 and $15.00. The Renville-Redwood Society is giving $60.00 in prizes for each county.

All societies and members that are interested in futhering the project are asked to write Dr. Cosgriff for further information and action.

NEW CANCER REGISTRY SYSTEM

An improved system for recording, indexing, filing and follow-up of cancer cases has been announced by the Physicians' Record Company, publishers of medical and hospital record forms. Adoption of a registry system is necessary for approval of the cancer program in a hospital or clinic.

Conforming to specifications in the *Manual of Cancer Registries and Cancer Clinical Activities* of the American College of Surgeons, this system of standardized forms provides a Cancer Accession Register, a means of indexing and filing cancer case records, and a follow-up control. Sites and histological types of tumors are classified in accordance with the *Standard Nomenclature of Diseases and Operations* to create a cancer index. Maintaining the system will demand a minimum of time from the medical record librarian. A new Cancer Registry Summary Sheet, a new Cancer Registry Index Folder and original colored plastic signals for indexing and follow-up are parts of the system.

For samples and detailed information, write to Physicians' Record Company, 161 West Harrison Street, Chicago 5, Illinois.

Committee Action

Committee on Syphilis and Social Diseases

Incidence of Venereal Disease
Tribute to Dr. Harry G. Irvine

In spite of experience to the contrary elsewhere in scattered spots over the country, in Minnesota there has been a continuing decrease in the incidence of syphilis and gonorrhea for many years, including 1956.

Incidence of Venereal Disease

A total of 196 cases of syphilis was reported in Minnesota for the year 1956; in 120 of them the patients were more than fifty years old, and twenty-seven were younger than thirty years. In these 196 cases, 139 patients were married, widowed, divorced or separated.

It is remarkable that in only twenty-one cases was the disease listed as primary or secondary. Among the other cases, the disease was early latent in nineteen, late or late latent in 151 and so-called congenital in five. Again this year, no baby was born in Minnesota with syphilis, a record which has been sustained for five of the last six years.

The total number of blood tests carried out for civilians was 197,728; for the armed services, 4,491. Specimens of cerebrospinal fluid tested numbered 3,939. Of twenty specimens submitted from genital lesions for darkfield examination, only two were found to contain the micro-organisms.

As pointed out previously by Dr. H. G. Irvine (who deplored the practice), physicians continue to institute treatment for suspected early lesions of syphilis without prior establishment of the diagnosis of that disease. Also, examination of the cerebrospinal fluid is not being carried out in enough cases.

The incidence of reported cases of gonorrhea was reduced from 1,000 in 1955 to 778 in 1956.

Tribute to Dr. Harry G. Irvine

It is high time that the Minnesota State Medical Association pay formal recognition to the extraordinary efforts towards venereal disease control in public-health work contributed by Dr. Harry G. Irvine, who has served as director of the Division of Venereal Disease of the Minnesota Department of Health since 1918. Dr. Irvine long ago recognized the importance of epidemiologic and follow-up studies and of direct contact with physicians in private practice through the efforts of social-service workers. Many of the state health laws and regulations were formulated under Dr. Irvine's direction, and are still effective. In 1936 Miss Lucy Clare Finley was brought in as executive assistant in charge of medical-social work in connection with venereal disease control. The recording of cases as reported by physicians and the follow-up studies have been conducted largely under the direction of Miss Finley and Dr. Irvine.

Minnesota throughout the years has enjoyed a high degree of freedom from venereal disease. Even during the war years the incidence of syphilis, based on reported tests, was less than 1 per cent.

In 1949 Dr. Irvine retired as clinical associate professor of dermatology and syphilology in the College of Medical Sciences at the University of Minnesota, and in 1953 he retired from active practice. For many years he served as chairman of this committee.

Dr. Irvine has at all times co-operated closely with physicians throughout the state in the investigation of venereal disease contacts, the reporting of cases and the follow-up of adequate treatment. As the picture of syphilis gradually changed after the introduction of penicillin, he has continued to advise physicians by mail and by personal contact. His contribution to public-health work in this field has been unequalled by anyone else in the states of the Union.

Louis A. Brunsting, M.D.,
Rochester, Minnesota,
Chairman

SOCIAL SECURITY

The government, while slow to acknowledge anything wrong with the Social Security System, underestimated the demand for benefits. Women who could obtain benefits at sixty-two, sixty-three, and sixty-four decided to do so even if the payments were less than they would be at sixty-five. Farmers suddenly turned out to be older than expected. Some began to pay social security taxes on reported income of $4200 which exceeded their income in prior years. Then they applied for benefits after paying taxes for six quarters. Many people who had retired and were well beyond sixty-five years of age, dug up jobs for themselves and paid social security taxes for eighteen months, thereby qualifying for benefits of from $30 to $108.50 monthly for life. Social security experts, in making their cost projections, underestimated the ingeniousness of the American people when Federal give-aways are as widely advertised as are social security benefits.

History of Medicine in Minnesota

NOTES ON THE MEDICAL HISTORY OF BECKER COUNTY

(Concluded)

G. G. HAIGHT, M.D.

Audubon, Minnesota

Dr. John Brainard Carman, one of the pioneer citizens of Detroit, was an 1879 graduate of McGill College, Montreal, Canada. Born in Iroquois, Ontario, he came directly to Detroit upon graduation; he received his license December 28, 1883. Dr. Carman devoted his life to the noble work of ministering to the sick and unfortunate. In every home he frequented, the announcement of his death was followed by a deep and genuine sorrow, for Dr. Carman had not only been the family physician; he had also been the advising friend, whose smiling face and cheery presence and word of confidence brought renewed hope to many homes which seemed threatened with disaster. He went about his work with little thought of self or of personal gain for more than thirty years. He made friends with all with whom he came in contact. In every relation to the community in which he lived, he was regarded by those who knew him best as a splendid example of the noblest work of God, an honest, manly man, a citizen of the highest type, and one whom it was deemed a privilege to call friend.

Dr. Carman died on September 13, 1913, at the age of fifty-eight. The attendance at the funeral was the largest ever seen in Detroit on a similar occasion. It was ironical that the doctor got a fishhook caught in his thumb and, like a doctor, thought not too much about it until it was too late. He had several sons and one daughter.

One son, **Dr. James Edwin Carman,** born in Iroquois, Ontario, on December 17, 1877, graduated from the Medical School at the University of Minnesota in 1901. After a very short time in Frazee, he joined his father and continued to practice in Detroit after the latter's death—with the exception of a short period around 1909 in Brooks, Red Lake County—until he retired in 1950 and removed to California.

Dr. John H. Carson graduated from McGill University Medical Department, Montreal, Canada, in 1881. He was licensed in Minnesota, November 14, 1883, and located in Lake Park. There he was listed in 1886 and 1890. He then moved to Duluth where directories list him from 1895 through 1909. Nothing more is known about him.

Dr. William E. Everley is listed in Polk's Medical Directory as practicing in Detroit from 1898 through 1902. Minnesota directories, however, list him there also in 1903 and 1905. It seems he did not graduate from a medical school. He was licensed by "1887 affidavit" on November 21, 1899 on the basis of his own statements that he was forty-two years old and that he had resided in Minnesota since 1864. He claimed that he started to practice as a student under the supervision of a preceptor, a Dr. William Riley, in Stearns County before 1880, that he had practiced also in Todd County and Becker County, had taken a course of lectures at the Department of Medicine of Northwestern University at Chicago in 1885 and submitted to the Board of Medical Examiners a certificate from this University which he received in 1890.

He must have been of a restless nature or perhaps had difficulty in establishing a good practice, because we find him in Twin Brooks, South Dakota, in 1886, in Grey Eagle, Todd County, in 1890 and 1895, in Glyndon, Clay County, in 1896, in Wadena, Wadena County, in 1898, and in Detroit, from 1900 through 1905. After that, nothing is known about him. He never was listed in any of the AMA directories.

Dr. John H. Froshaug* apparently was of Scandinavian descent, but nothing can be found about his early or medical education. He practiced in Albert Lea, Freeborn County, from 1871 to 1877, though the *Enterprise* mentions his imminent departure in 1876. He was associated in the drug business there with Dr. Frank A. Blackauer. In July, 1877, he moved to Becker County, locating in Lake Park. In 1879, he was elected a delegate for Becker County to the Republican State Convention. He was coroner from 1880 to January, 1882.

Later, Dr. Froshaug moved to Twin Valley, Norman County, where he is listed in 1886. He died there on June 20, 1889.

Dr. Rowland Thomas S. Gilmore came to Lake Park in September, 1896, and remained there until December, 1899, when he left for Chicago to do postgraduate work.

Lake Park at that time had a population of about 400 and the only doctor was Dr. O. K. Winberg.

Dr. Gilmore was born in Toronto, Canada, December 12, 1869, and graduated from Trinity Medical College in 1895. He was licensed in Minnesota, October 13, 1896, by examination, though he was engaged by the Northern Pacific Benefit Association a year previously (in October, 1895) and he worked on the staff of the sanatorium of this organization at Brainerd until he moved to Lake Park.

He returned to Minnesota from Chicago in 1900, settling in Fosston, Polk County, where with a former classmate, Dr. Malcolm MacKinnon, he established a hospital.

In 1903, he moved to Bemidji, Beltrami County, where he stopped practicing December 14, 1947, because of illness. He went first to St. Vincent's Hospital at Crookston and in 1952 to the Rodwell Rest Home at Mahnomen, where he is still staying (1956).

Dr. Knute K. Halvorson practiced in Audubon from 1889 to 1913. He graduated from Hamline University in 1889, but was not properly licensed. He got into difficulties with the Board of Medical Examiners because he practiced without a license and because he supposedly practiced under a different name.

He was born in Norway on February 2, 1855. According to some information, he graduated from the Medical School of Hamline University in 1899. It is said that he returned to Norway in 1915.

Dr. Christian J. B. Hirsch (or Hursch) graduated from Rush Medical College, Chicago, in 1868. At first, he practiced in Black River Falls, Wisconsin. It is not certain when he moved to Minnesota, but he was licensed here in 1884 and he practiced in New Ulm, Brown County, in 1891. In 1899, he came to Audubon where he developed a large Scandinavian practice. He died in Audubon on October 16, 1901.

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*See Medical History of Freeborn County, MINNESOTA MEDICINE, December, 1948.

Dr. Joseph G. Hodgkinson is listed in Detroit in 1886 and 1890. He may have been there before 1886, as he was licensed by examination on October 12, 1883, apparently three years before he graduated from the St. Paul Medical College (1886). In 1893, he was in Osage. In 1895 and 1898, we find him listed as practicing in Menagha, Wadena County, and from 1903 through 1909 in South Saint Paul. It is not known what happened to him after that.

Dr. Edward E. Hoit was born in Auburn, Maine, September 3, 1854. He graduated from the University of Michigan Homeopathic Medical College in 1878, from the University of Michigan Department of Medicine and Surgery in 1878, and from Rush Medical College, Chicago, in 1885. He is listed in Detroit from 1889 on.

He married Della Day, October 24, 1889, and one married daughter still lives at Detroit Lakes, as the town is now called. He was county physician from 1884 to 1887, from 1889 to 1891, and from 1895 to 1903.

Dr. Hoit was a very small man, but a courtly gentleman with a large fund of stories, some risque, some otherwise. While Dr. J. B. Carman rode a bicycle to see his patients, Dr. Hoit was very partial to livery horses, and Dr. Weeks was one of the first to take up with the new cars. Dr. Hoit died at Detroit Lakes, April 4, 1925.

Dr. Thomas Howell, a graduate of Dartmouth Medical College in 1896, is listed in Frazee in 1898 when this town had a population of 279.

Dr. Samuel S. Jones was one of the pioneer physicians at Frazee. He was born in New York on February 23, 1849. His medical education was received at Rush Medical College, Chicago, from which he graduated in 1881. He was licensed in Minnesota by diploma on November 24, 1883. At first, he located in Minneapolis where he is listed as early as 1890. It seems he came to Frazee in 1897, as he was coroner there in that year, an office he held until 1899. He remained in Frazee until at least 1927, when he retired and moved to Minnneapolis. He died in Minneapolis on November 12, 1934, at the age of eighty-five, from coronary occlusion and infarct.

Dr. Niles (also Nils and Miles) R. H. Juell (or Jewell) is listed in Lake Park in 1886 and 1890. He had graduated from Rush Medical College in Chicago in 1884 and was licensed in Minnesota, September 26, 1885. In 1895 and 1898, he is listed in Red Wing, Goodhue County, and in 1903 and 1905 in Benson, Swift County. In 1909 or before, he moved to Santa Rosa, California, where he was as late as 1927.

Dr. C. A. Lampanius was of Finnish descent. He was coroner of Audubon from January, 1876, to January, 1880. He graduated from the College of Physicians and Surgeons, Keokuk, Iowa, in 1878 and was licensed October 23, 1883. He therefore, started to practice before his graduation. He is listed in Audubon as late as 1890. From at least 1895 through 1905, he was in New York; in 1909 in Catonsville, Maryland. Nothing further is known of him.

There is a possibility that his wife, too, practiced medicine, but no record of her could be found.

A **Dr. Lieman** is listed in Frazee in 1886. At that time, Frazee was a small village of 150 people. He was not licensed in Minnesota and no further information could be discovered.

Dr. Dexter J. Maltby was the first civilian physician to settle in Becker County. He was born in Watertown, New York, on April 25, 1843. During the Civil War, he served in a New York regiment. For a time, he was a prisoner of war and then returned to Watertown where he studied and received a certificate from the examining board in 1871. He settled in Detroit in April of the same year when this town was still quite new. He died in Detroit, June 8, 1880.

Dr. M. Morton, a physician of the "regular" school, is listed in Detroit in 1893. He was coroner from 1889 to 1903. Nothing more is known of him. (A Dr. Howard McIlvain Morton is listed in the records of the Minnesota State Board of Medical Examiners, but the list does not show him in Becker County.)

Dr. Aretus K. Norton graduated from the Chicago Medical College in 1872. It is not known when he came to Detroit where he is listed as practicing in 1886 and 1890. He was licensed October 11, 1883. From about 1895 to 1905, he was in Minneapolis and in 1909 in Duluth.

Dr. Emma Katherine Ogden practiced in Detroit for thirty-five years. She was born in Pittsburgh, Pennsylvania, on February 21, 1840, and graduated from the Woman's Medical College of Philadelphia in 1875. She was for a time a missionary of the American Board of Madura, India. She was licensed in Minnesota November 12, 1883. If it is correct that she practiced in Detroit thirty-five years, she must have located there about 1881. Dr. Ogden owned a drug store at Detroit and was vice president of the State Pharmaceutical Society. She died at Detroit on April 5, 1916, from pneumonia.

Dr. W. Thornton Parker graduated from the Royal University of Munich. For some time he resided in Boston, Massachusetts, and was surgeon at White Earth for a time before moving to Winona in May, 1881.

Dr. David Pyle was born in Ohio about 1825. In Minnesota, he settled first in McLeod County and became a member of the legislature in 1868. In the following year, he was appointed government physician at White Earth. In 1870, he settled on a claim in Audubon. He advised the early settlers to stick to their claims; he would warn the Indians at the Reservation that the settlers were armed and would fight for their rights. The Indians were displeased with the use of their hunting grounds for farming. Many of the early settlers stopped with Dr. Pyle until they could get some shelter put up on their claims. It is said he never turned anyone away as long as there was room for a bedroll in his home.

He obtained a commission as notary public for Alexandria, Douglas County. When Becker County was organized, he was appointed the first County Auditor, and in 1871 he was elected County Coroner. He must have been very popular because when he was to move to Monticello the pioneer settlers gave a banquet in his honor and presented him with a gold headed cane on which a chronicle of his helpful deeds to the settlers was engraved. He moved to Alabama in 1873 and died there about 1884.

Dr. M. B. Smith was born on a farm near Hamilton, Ontario, in the year 1868. He graduated in medicine at the University of Toronto and started to practice medicine with an uncle at Wabashene, Ontario. He next practiced at Hagersville, Ontario, for three years. He then took and passed the State Board examination of the State of Michigan. In the early 1900's, he and his wife settled in Lake Park.

After selling his practice to Dr. R. M. Gunderson, he made insurance his main work. After fifteen years in Lake Park, he moved to Ottumwa, Iowa. Dr. Smith was a large man, resembling in appearance the late Charles Evans Hughes. He was very well liked and got along well with everyone. He died at Ottumwa in 1934.

Dr. Paul Sorkness was listed in Lake Park in 1898. He apparently moved there from Fisher, Polk County, where he practiced around 1896. Later, he moved to Fargo, North Dakota, where he practiced from at least 1903 on.

Dr. Sorkness graduated from the College of Medicine and Surgery of the Unisity of Minnesota in 1895 and was licensed in the same year. While in Fargo, he was very active in North Dakota medical affairs. He was president of the State Medical Society, also of the State Board. He was also a railroad surgeon.

He died in Fargo on September 23, 1920, from cerebral hemorrhage, at the age of fifty-three years.

Dr. Byron F. Van Valkenberg was in Becker County in 1895. He was born in Marquette, Wisconsin, in April, 1864, and graduated from a medical school in 1893. He received his Minnesota license on July 7 of the same year. From Becker County, he moved to Long Prairie, Todd County, where he practiced many years until he retired.

Dr. Leonard Case Weeks was born in Hartland, Wisconsin, in 1869. Not much is known about his boyhood. He graduated from Rush Medical College in 1892, interned at the Presbyterian Hospital, and then taught Physiology and Anatomy at Rush. Dr. Weeks commenced the practice of medicine in Detroit in 1896, though he had been licensed in Minnesota in 1892. He maintained the Detroit Hospital from 1905 to 1926, with the exception of five years of this period when it was operated by nurses.

Dr. Weeks had the same large territory that most country doctors tried to cover in those early days, mostly with horses and buggy or sled. He soon was doing most of the surgery in the county and, for a country doctor, had a good bent in this direction.

Becker County was first settled by people of the same nationality as Dr. Weeks, but soon the Scandinavians began to come across the ocean, mostly through Quebec or Halifax and some through New York City. Dr. Weeks was interested in the Scandinavian language and became quite proficient in reading it. He also was interested in genealogy and traced his ancestors down from colonial times.

Dr. Weeks held many public offices, such as coroner and county physician. He was health officer from 1911 to 1925 (part of this time chief health officer) and mayor from 1909 to 1911. For nineteen years, he served as school director. He was a member of the Minnesota State Medical Society and the Clay-Becker County Medical Society. For some time he was examiner of the John Hancock Mutual Life Insurance Company, the MWA, RNA and AOUW.

After a quarter of a century of putting up with long drives over bad roads, cold winters, long hours at night, loss of sleep, disappointments, and, yes, the memory of mistakes and shortcomings, the doctor began to show the effects of these things. People began to call him "Old Doc," and looking in the mirror, he saw that something had made his hair white before his time. He was growing heavier, his cheeks looked a little too full, and, alas, he had a "bay window."

While the doctor was sitting in his office one day he felt a numbness in his left hand. The hand and arm were lying on his left thight and he noticed that there was no pressure on the arm to make it numb. Then he moved the left foot and found it numb, too. The combination was suggestive, so he stood up and walked across the floor. He noticed that the left foot dragged and the toe of his shoe scuffed along the carpet and that he was unsteady in his walk. "Old Doc" had contemplated some such emergency. A somewhat radical increase in blood pressure, breathlessness on exertion, some rather hard beating of his heart, and a suspicious faintness the preceding summer had given him warning. He walked to the phone to call a friendly doctor and asked a friend to bring his car down to the office to take him home. His wife met him at the door and ushered him in with her usual calmness, for the doctor and his wife were not given to expressions of emotion, at least not outwardly. He went to bed and in a few days it was decided that a long rest was necessary. The doctor and his wife packed up and took a trip to the West Coast, a trip which they had long hoped for but under entirely different conditions. After three months of traveling through the West, "Old Doc" began to gather strength and he and his wife returned to what they called home. He was tired but went to work. Perhaps the doctor was too ambitious, but it must be admitted that it seemed as if too many of his old clients looked the other way when they met. While attending a football game in Minneapolis, he suffered another stroke and in a few weeks died at Detroit on December 19, 1926, at the age of fifty-seven years, nine months.

Dr. B. Dudley Williams graduated from the University of Louisville Medical Department in 1884. He is listed in White Earth in 1896. There is no record of his being licensed in Minnesota, nor any other information.

Dr. Oesteu K. Winberg was born in Nordfjordeid, Norway, July 20, 1864. He came to the United States when seventeen years old, graduated from Augsburg College, and completed the course in medicine at the University of Minnesota in 1892. He was licensed on April 7, 1893, by examination.

After practicing at Willmar, Minnesota, for one year, he came to Lake Park, where he practiced until his health forced him to give up his life work.

He married Alpha Carlson, who was an ideal wife and physician's helpmate. When one of the children swallowed a piece of silver money, he said, "Mother, what shall we do?" She merely turned the boy upside down, hit him on the back, and out rolled the money.

Dr. Winberg spent the year 1902 studying in Germany and visiting in Norway. He returned again in 1910 when he gave 1,000 Kr. to a hospital in Eid, Norway and 1,000 Kr. to a forest development. Upon returning to this country, he took postgraduate work in New York.

He was very much interested in cancer. One of his prized possessions was a cane given to him by the Minnesota State Medical Society. He was the active organizer of the movement that created the Sand Beach Sanatorium and was one of the directors for twelve years. He was president of the school board at the time of his death, December 27, 1930. He was buried on his thirty-fifth wedding anniversary. His practice was taken over by Dr. R. V. Jolin.

Acknowledgment

We wish to acknowledge help from many sources, especially that of Dr. R. Rosenthal. Without his aid, many doctors would have been overlooked.

Meetings and Announcements

STATE

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

NATIONAL

Alumni Post Graduate Medical Convention, College of Medical Evangelists, twenty-sixth annual meeting, February 25-27, Hotel Biltmore, Los Angeles, California.

American College of Surgeons, sectional meeting, Des Moines, Iowa, March 27-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

Mediclinics of Minnesota. Fort Lauderdale, Florida, March 2-12, 1958.

Oklahoma Colloquy on Advances in Medicine, first meeting, February 6, 7 and 8, 1958, University of Oklahoma School of Medicine, Oklahoma City, Oklahoma.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

International Society of Internal Medicine, Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

World Congress of Gastroenterology, Washington, D. C., May 25-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

SCIENTIFIC EXHIBITS SOLICITED

Members of the Minnesota State Medical Association are invited to exhibit a scientific project at the state meeting to be held May 22, 23, and 24, 1958 at the Minneapolis Auditorium, preceding the tenth anniversary meeting of the World Health Organization in Minneapolis. Treatment and management of special problems, better ways to do procedures, slants or tips on office management are several suggested ideas. Space will be available without charge. Mail exhibit space requests to Dr. Arnold Kremen, Chairman for Scientific Exhibits, Local Arrangements Committee, Minnesota State Medical Association, 496 Lowry Medical Arts Building, St. Paul 2, Minnesota.

MEDICAL LIBRARY ASSOCIATION

Medical Library Association's fifty-seventh annual meeting will be held in Rochester, Minnesota, June 2 through June 6, 1958, with headquarters at the Hotel Kahler. The theme of the Rochester meeting will be "Advances in Medical Library Practice."

POSTGRADUATE COURSE ON DISEASES OF THE CHEST

The Council on Postgraduate Medical Education of the American College of Chest Physicians will sponsor the 11th Annual Postgraduate Course on Diseases of the Chest at the Warwick Hotel, Philadelphia, March 3-7, 1958. The tuition fee is $75.00 including round table luncheons. Further information may be obtained by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

CONTINUATION COURSES

Medical continuation courses to be presented at the Center for Continuation Study, University of Minnesota, are as follows:

January 30-February 1	Emergency Surgery for General Physicians
February 6-8	Cardiovascular Diseases for General Physicians
February 10-15	Neurology for General Physicians
March 3-5	Pediatrics for General Physicians
March 17-19	Internal Medicine for Internists
March 20-22	Surgery for Surgeons

For further information concerning the above courses, write to the Director, Department of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.

METHODIST MISSION MEDICAL NEEDS CITED

The Board of Missions of the Methodist Church has announced a need for twenty doctors in its mission fields in ten countries overseas in 1958.

The board, through its Office of Missionary Personnel, is seeking to recruit ten women and ten men to serve as medical missionaries. The openings cover a varied range of medical fields, including internal medicine, surgery, general practice, public health, gynecology, pathology and radiology.

In 1958, doctors are being sought for work in the Belgian Congo, Mozambique, Southern Rhodesia, Angola, India, Pakistan, Korea, the Philippines, Sarawak (Borneo), and Mexico.

Persons interested in obtaining information about medical missionary service may write: Office of Missionary Personnel, 150 Fifth Ave., New York 11, New York.

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AMA House of Delegates Holds Eleventh Clinical Meeting

Dr. David B. Allman, president of the American Medical Association, called for "more freedom, not less, in America and the medical profession," in his address to the opening session of the Eleventh Clinical Meeting held December 3-6 in Philadelphia.

Dr. Allman urged the delegates to embark on local action campaigns to enlist full community support in opposition to the Forand Bill, a pending Congressional proposal which would provide hospital and surgical benefits for persons who are receiving or are eligible for Social Security retirement and survivorship payments. The Forand Bill, he said, is "cut from the same cloth" as national compulsory health insurance and "emanates from the same minds."

Fluoridation of Water

The House of Delegates approved a joint report of the Council on Drugs and the Council on Foods and Nutrition which endorsed the fluoridation of public water supplies as a safe and practical method of reducing the incidence of dental caries during childhood. The report contained these conclusions:

1. Fluoridation of public water supplies so as to provide the approximate equivalent of 1 ppm of fluorine in drinking water has been established as a method for reducing dental caries in children up to 10 years of age. In localities with warm climates, or where for other reasons the ingestion of water or other sources of considerable fluorine content is high, a lower concentration of fluoride is advisable. 2. No evidence has been found since the 1951 statement by the Councils to prove that continuous ingestion of water containing the equivalent of approximately 1 ppm of fluorine for long periods by large segments of the population is harmful to the general health. Mottling of the tooth enamel (dental fluorosis) associated with this level of fluoridation is minimal. 3. Fluoridation of public water supplies should be regarded as a prophylactic measure for reducing tooth decay at the community level and is applicable where the water supply contains less than the equivalent of 1 ppm of fluorine.

Free Choice of Physician

The House passed a resolution which reaffirmed approval of previous interpretations of the Principles of Medical Ethics by the Association's Judicial Council and directed that they be called to the attention of all constituent associations and component societies. One Council opinion, issued in 1927 and reaffirmed in Philadelphia, stated that the contract practice of medicine would be determined to be unethical if "a reasonable degree of free choice of physician is denied those cared for in a community where other competent physicians are readily available." The resolution also cited a Council opinion, published in the October 19, 1957, issue of *The Journal of the A.M.A.*, which stated that the basic ethical concepts in both the 1955 and 1957 editions of the Principles of Medical Ethics are identical in spite of changes in format and wording. The 1927 Council report also pointed out that "there are many conditions under which contract practice is not only legitimate and ethical, but in fact the only way in which competent medical service can be provided." Judgment of whether or not a contract is ethical, the report said. must be based on the form and terms of the contract as well as the circumstances under which it is made.

The Heller Report

Acting on the report of the Committee to Study the Heller Report on Organization of the American Medical Association, the House reached the following decisions on ten specific recommendations:

1. The office of Vice President will be continued as an elective office.

2. The offices of Secretary and Treasurer will be combined into one office to be known as Secretary-Treasurer, and that officer will be selected by the Board of Trustees from one of its number.

3. The duties of the Secretary-Treasurer will be separated from those of the Executive Vice-President.

4. The office of General Manager will be discontinued, and the new office of Executive Vice President will be established. The latter, appointed by the Board of Trustees, will be the chief staff executive of the Association.

5. The Council on Medical Education and Hospitals and the Council on Medical Service will continue as standing committees of the House of Delegates, but their administrative direction will be vested in the Executive Vice President.

6. The voting members of the Board of Trustees will be limited to eleven—the nine elected Trustees, the President and the President-Elect. The Vice President and the Speaker and Vice Speaker of the House of Delegates will attend all Board meetings, including executive sessions, with the right of discussion but without the right to vote.

7. The House disapproved of the proposal to elect the Trustees from each of nine physician-population regions.

8. The office of Assistant Secretary will be discontinued, and a new office of Assistant Executive Vice-President will be established.

9. The Committee on Federal Medical Services will be retained as a committee of the Council on Medical

(Continued on Page A-6)

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Service and will not become a part of the Council on National Defense.

10. The Speaker of the House will appoint a joint and continuing committee of six members, three from the Board of Trustees and three from the House, to redefine the central concept of AMA objectives and basic programs, consider the placing of greater emphasis on scientific activities, take the lead in creating more cohesion among national medical societies and study socio-economic problems.

The Forand Bill

The House condemned the Forand Bill as undesirable legislation, approved the firm position taken in opposition to it and expressed satisfaction that the Board of Trustees has appointed a special task force which is taking action to defeat the bill.

Health Programs for Hospital Employees

A set of "Guiding Principles for an Occupational Health Program in a Hospital Employe Group" was approved by the House. The guides were developed by a joint committee of the American Medical Association and the American Hospital Association and already had been formally approved by the A.H.A.

Asian Influenza Vaccine

The House considered three resolutions dealing with the Asian influenza immunization program and then adopted a substitute resolution calling attention to "certain inadequacies and confusions in the distribution of vaccines" and directing the Board of Trustees to seek conferences through existing committees "with a view to establishing a code of practices regulating the future distribution of important therapeutic products, so that the best interest of all the people may be served."

Medical Rating of Physical Impairment

The House accepted a 115-page "Guide to the Permanent Impairment of the Extremities and Back" which was developed by the Committee on Medical Rating of Physical Impairment as the first in a projected series of guides.

Miscellaneous Actions

Among a wide variety of other actions, the House also: Affirmed that it is within the limits of ethical propriety for physicians to join together as partnerships, associations or other lawful groups provided that the ownership and management of the affairs thereof remain in the hands of licensed physicians; Instructed that the appropriate committee or council should engage in conferences with third parties to develop general principles and policies which may be applied to the relationship between third parties and members of the medical profession; Asked the Board of Trustees to study the feasibility of having the Association finance a thorough investigation of the Social Security system by a qualified private agency; Suggested that physicians and their friends make a vigorous effort to obtain Congressional enactment of the Jenkins-Keogh Bills; Approved the "Suggested Guides to Relationships Between Medical Societies and Voluntary Health Agencies."

STUDY ON CIGARETTE SMOKING

Tobacco company employes smoke more, but have a lower over-all death rate than the general population, a lower death rate for cancer generally, for cardiovascular diseases, and for coronary diseases, and a respiratory cancer death rate no higher than that of the general population. Harvey B. Haag, M.D., and Hiram R. Hanmer of the Department of Pharmacology of the Medical College of Virginia, report a study correlating smoking habits with the mortality rate of employes of nine processing plants of the American Tobacco Company, in an article in the December, 1957, issue of *Industrial Medicine and Surgery.*

A study made in 1956 showed that the proportion of tobacco company employes consuming more than twenty cigarettes daily was double that of the general population. In all age groups, male and female, white and nonwhite, there was a higher percentage of smokers and a higher daily consumption of cigarettes among the tobacco company employes than in the general population.

Based on the death rates for the general population of the United States, the authors computed the "expected" number of deaths in the employe population and compared them with the number that actually occurred for the years 1953-1956. The entire group of factory workers represented 45,455 person-years of exposure during the period studied.

The expected number of deaths and the number actually observed were reported as follows: deaths from all causes—350 expected, 241 observed; deaths from all forms of cancer—61 expected, 47 observed; deaths from respiratory cancer—10 expected, 10 observed; deaths from cancer of trachea, bronchus and lung—4 expected, 4 observed; deaths from cardiovascular diseases—137 expected, 104 observed; deaths from coronary diseases—82 expected, 56 observed.

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License of Former Fairfax Physician Suspended for Five Years

On November 8, 1957 the Minnesota State Board of Medical Examiners suspended for a period of five years the medical license held by Dr. Arthur T. Farisy, 65 years of age, who for the past several years has been engaged in the practice of medicine at 3200 W. National Ave., Milwaukee, Wisconsin. Dr. Farisy had been charged in a citation issued by the Board with having been convicted of a felony. Dr. Farisy was not present at the hearing due to the fact that on August 23, 1957 he had been sentenced by the Hon. Herbert J. Steffes, Judge of the Municipal Court of Milwaukee, Wisconsin, to two concurrent terms of not more than three years each in the Wisconsin State Prison at Waupun, Wisconsin, for supplying narcotic prescriptions to addicts. Dr. Farisy had entered a plea of guilty to two criminal offenses, a breach of good faith in prescribing narcotic drugs and unlawfully prescribing narcotic drugs.

Dr. Farisy was arrested following an investigation which began as the result of a raid made by Milwaukee police officers on August 18, 1957 in which several thousand dollars worth of narcotics and narcotic equipment were seized and three Chinese men were arrested. According to a Milwaukee detective, the men admitted obtaining some of the drugs from a doctor's prescription and the prescriptions written by Dr. Farisy were then uncovered in searching the records of various drug stores. It was found by the police that Dr. Farisy had issued 112 prescriptions for dolophine for one of these Chinese "patient" from January 9th to August 16th, out of a total of approximately 450 similar prescriptions written by the doctor in the same period. Dr. Farisy admitted that he received between two dollars and five dollars for each prescription and also that he postdated many prescriptions which were filled when he left town.

Dr. Farisy, who was born in Franklin, Minnesota, on August 24, 1892, graduated from Rush Medical College in 1920. He was licensed to practice medicine in Minnesota in 1923 by reciprocity and practiced for a number of years at Fairfax, Minnesota, before moving to Wisconsin.

License of St. Paul Physician Revoked for Claims in Treatment of Cancer

On November 8, 1957 the Minnesota State Board of Medical Examiners revoked the medical license held by Dr. Herman A. Hartung, eighty-four years of age, who formerly maintained an office for the practice of medicine at 321 First Federal Bldg., St. Paul, Minnesota. Dr. Hartung had been charged by the Board in a citation issued on July 25, 1957 with immoral, dishonorable and unprofessional conduct as defined by law in that he had offered in writing to accept a fee in the sum of $100 from a patient who was afflicted with cancer of the sigmoid colon, with metastasis to the liver and lungs, for a treatment that involved the use of "chemicals and drugs in the form of a synthetic antitoxin which is injected into the muscle tissue."

Dr. Hartung was further charged with stating in the letter which was mailed to the wife of the patient, that he had a test which showed what stage of advancement the cancer was in and that "many cases in the first or second stage often get well with only one shot of the antitoxin. The farther advanced cases naturally need more." In his letter Dr. Hartung urged the wife to bring her husband to his office. Other statements in the letter termed false and misleading by the Board were as follows: "With the old method of treating by surgery and x-ray, the patient really has no chance whatever. My record shows, that taking them as they come, I cure better than sixty-five per cent. If I would pick my cases I could or would have a ninety to ninety-five per cent cures." Upon being questioned by representatives of the Minnesota State Board of Medical Examiners, Dr. Hartung admitted that he had no records and therefore could not substantiate his claim of curing so many patients.

Although Dr. Hartung did not appear at the hearings before the Board either on August 23, 1957, or on November 8, 1957, he had admitted previously to representatives of the Board that he had administered the Koch oxidation catalyst treatment over a period of many years to patients who were suffering from cancer. According to Dr. Hartung, the liquid which was injected into the patient contained a chemical called "glyoxylide." He also said that he charged the patient $100 for each injection out of which he paid $25 to a Detroit, Michigan, firm for a vial of the preparation, but Dr. Hartung said the $100 charge also included his fee for the examination. In reference to his test for cancer, Dr. Hartung stated in a letter sent by him to the Board: "I built my method of early detection and determining the stage of advancement of cancer on the pioneer work by Dr. Geo. S. White known as the Bio-Dynamo-Chromatic vagal reflex test. With the proper technique, this has given me almost unerring accuracy on the condition on hand."

After the testimony of the witnesses had been taken at the hearing on August 23, 1957, the attention of the Minnesota State Board of Medical Examiners was called to the fact that in the case of Koch et al vs. Federal Trade Commission, the United States Court of Appeals for the Sixth Circuit had held in an opinion dated July 8, 1953, that the evidence sustained a finding by the Commission that representations made in advertising concerning certain products, one of which was "Glyoxylide," were false and misleading in material matters and that the products involved had no therapeutic value. The complaint in the case brought by the Federal Trade Commission alleged that Koch Laboratories, Inc., and petitioners William F. Koch and Louis G. Koch, President and Secretary-Treasurer, respectively, of Koch Laboratories, Inc., were engaged in the manufacture and in the sale and distribution in interstate commerce of certain medicinal preparations designated as "Glyoxylide," "B-Q," and "Malonide Ketene Solution" and that, in the course and conduct of their business, petitioners William F. Koch and Louis G. Koch disseminated and caused to be disseminated, by the United States mails and by various means in commerce, false, misleading and deceptive statements and representations as to the therapeutic value of their aforesaid medicinal preparations for the purpose of inducing the purchase of these preparations.

It was also alleged in the complaint by the Federal Trade Commission that through the use of these statements and representations it was represented directly or by implication that "Glyoxylide" is an adequate treatment for any type or stage of cancer, leprosy, malaria, coronary occlusion or thombosis, multiple sclerosis, arteriosclerosis, angioneurotic edema, obliterative endarteritis, asthma, hay fever, dementia praecox, epilepsy, psoriasis, poliomyelitis, tuberculosis, syphilis, arthritis, and osteomyelitis, any type of allergy or infection, abscess of the prostate gland, septicemia, and insanity. When the case was originally heard before the Federal Trade Commission, thirty-three physicians and scientists testified for the Commission to the effect that the petitioners' product had no therapeutic value. Thirty-six witnesses, including twenty-nine physicians, testified on behalf of the petitioners.

Dr. Hartung was born in Washington County, Minnesota, on February 19, 1873, and graduated from the University of Minnesota with an M.D. degree in 1900, being licensed to practice medicine in Minnesota in June of that year. Dr. Hartung was engaged in the practice of medicine at Le Sueur, Minnesota, from 1906 until 1924 when he moved to St. Paul where he practiced until recently.

MEETINGS AND ANNOUNCEMENTS

(Continued from Page 68)

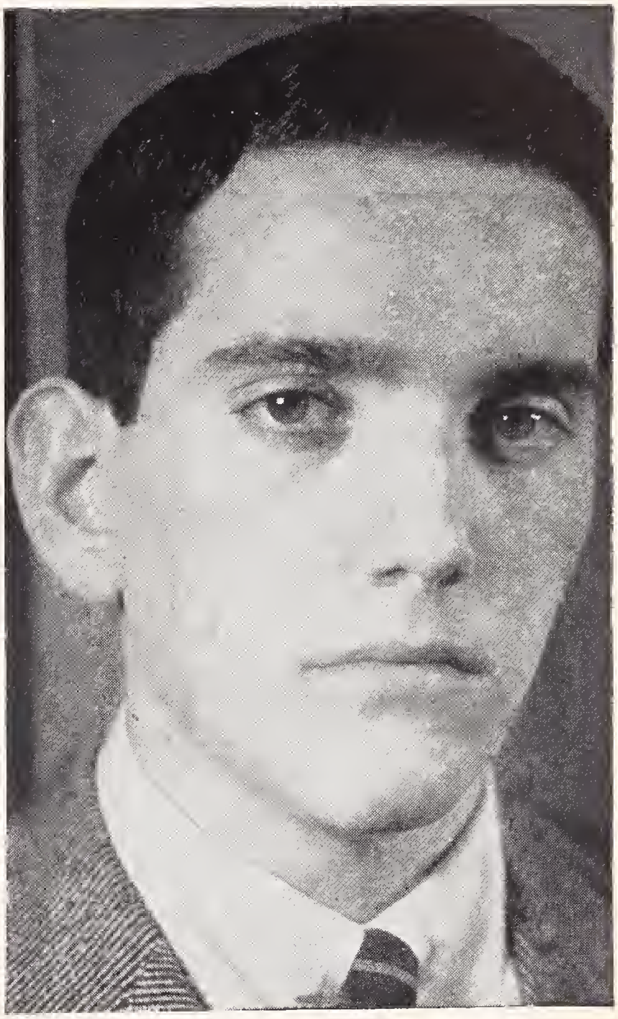
MISSISSIPPI VALLEY MEDICAL SOCIETY ESSAY CONTEST FOR 1958

The Mississippi Valley Medical Society has announced a cash prize of $100.00 for the best essay submitted in its 1958 essay contest. Any subject of general medical or surgical interest including medical economics and education may be submitted providing the paper is unpublished and is of interest and applicable value to general practitioners of medicine. Contributions are accepted only from physicians who are members of the AMA and who are residents and citizens of the United States. Manuscripts must not exceed 5,000 words and be submitted in five complete copies, in manuscript style. The winning essay receives a cash prize of $100.00, a gold medal, and a certificate, also an invitation to address the annual meeting of the Mississippi Valley Medical Society. (Held at time and place of the American Medical Writers' Association; 1958 Meeting, Hotel Morrison, Chicago, September 24, 25, 26.) The Society may also award certificates of merit to physicians whose essays rate second and third best. Essays must be in the office of the M.V.M.S. Secretary, Harold Swanberg, B.S., M.D., F.A.C.P., 209-224 W.C.U. Building, Quincy, Illinois, not later than May 1, 1958.

NATIONAL HEART INSTITUTE BOOKLET RELEASED

The U. S. Public Health Service has released Publication No. 513, entitled "Cerebral Vascular Diseases and Strokes," third ranking cause of death in the United States. A free copy may be obtained from the Heart Information Center, National Heart Institute, Bethesda 14, Maryland. The booklet, a publication of the Public Health Service's National Heart Institute, states that strokes and other effects of cerebral vascular disease caused an estimated 179,110 deaths in 1956, and that approximately two million people are incapacitated or handicapped by brain blood vessel disease.

Superior for acne cleansing



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In Memoriam

LLOYD L. MERRIMAN

Dr. Lloyd L. Merriman, Duluth physician specializing in internal medicine, died November 22, 1957 at his residence. He was sixty-seven years of age.

Dr. Merriman had practiced medicine in Duluth since 1919 and was a former chief of staff of St. Luke's Hospital and also was a member of the medical staff of St. Mary's Hospital.

Dr. Merriman was born in Huron, South Dakota. He studied at Cornell University, the University of Chicago and Rush Medical School, Chicago, receiving his M.D. degree from the latter institution in 1918. The following year he completed a fellowship for his specialty with Dr. B. W. Sippy in Chicago.

Active in Masonic affairs, he was a past master of Glen Avon Lodge, AF&AM, a past patron of the Glen Avon Chapter of the Order of Eastern Star and was a member of the Commandery and Aad temple of the Shrine.

Medical organizations in which he held membership included the St. Louis County Medical Society, the Minnesota State Medical Association, the American Medical Association, and the Minnesota State Mental Health Committee.

Surviving are his wife, Mamie; one son, Dr. Lloyd A., Marietta, Georgia; one daughter, Mrs. John Runquist, Duluth; two sisters, Mrs. Floyd Eberline, and Mrs. Henry Wallace, Britton, South Dakota; one brother, Hollis, Seattle, Washington, and eight grandchildren.

NESMITH P. NELSON

Dr. Nesmith P. Nelson, Minneapolis, retired physician and surgeon, died December 14, 1957, at the age of eighty years.

Dr. Nelson had practiced medicine in Minneapolis and Brainerd until the time of his retirement in 1953. He was an eye, ear, nose and throat specialist.

The deceased was born in Anoka, Minnesota, and his preliminary education was obtained at Minneapolis South High School. He received his medical degree at George Washington University in Washington, D. C. He did postgraduate work for one year at Manhattan Eye, Ear, and Throat Hospital, New York City, and also at Cornell University Medical School and New York Eye and Ear Infirmary. In 1920, he came to Minneapolis for a year, then moved to Brainerd until 1949.

He was a member of the Upper Mississippi Medical Society, an associate member of the Minnesota State Medical Association and a member of the American Medical Association. Other positions and professional affiliations held by Dr. Nelson included: Association of Military Surgeons of United States, George Washington University Medical Society and American Association of Ophthalmology. Dr. Nelson was also past president of Upper Mississippi Medical Society and a member of Zuhrah Temple, Scottish Rite and Sons of Norway.

Survivors include his wife, Rose, and two sons, Floyd A., Tacoma, Washington, and Russell D., Excelsior.

JOSEPH M. SPRAFKA

Dr. Joseph M. Sprafka, St. Paul surgeon, died December 6, 1957 in St. Joseph's Hospital after a lengthy illness. He was sixty-two years of age.

Born in Perham, Minnesota, he attended the public schools of that community. He was a graduate of the University of Minnesota Medical School, class of 1919. His internship included eighteen months at St. Anthony De Padua Hospital in Chicago, Illinois.

Dr. Sprafka was a resident of St. Paul for thirty-six years. He was an active member of the Minnesota State Medical Association, the Ramsey County Medical Society, and the American Medical Association. He was also a member of the International College of Surgeons.

Survivors include his wife, Bernice; three sons, Dr. Joseph L., Dr. Gregory A., and Robert F., all of St. Paul; two brothers, Dr. Ambrose, Detroit Lakes and Alfred, St. Louis Park; and eight grandchildren.

OSWALD S. WYATT

Dr. Oswald S. Wyatt, Minneapolis pediatric surgeon, died November 21, 1957 in Abbott Hospital in Minneapolis. He was sixty-three years old.

Dr. Wyatt was chief of children's surgery at Minneapolis General Hospital. In addition, he held the following positions and professiontl affiliations: assistant professor of surgery, University of Minnesota; consultant in surgical pediatrics, Minneapolis General Hospital; Glen Lake Sanatorium, and Lymanhurst Pediatric Hospital.

Born in Canada, he received his preliminary education at East High School in Minneapolis. In 1919, Dr. Wyatt was graduated from the University of Minnesota Medical School. He interned for one year at General Hospital and was the first resident in surgery to be trained there. In 1928, he became one of the first three men in the United States to begin specializing in pediatric surgery.

The deceased was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Survivors include: one son, Oswald S. Wyatt, Jr., Minneapolis; and a brother, Dr. Alvin S. Wyatt, Clinton, Wisconsin.

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* * *

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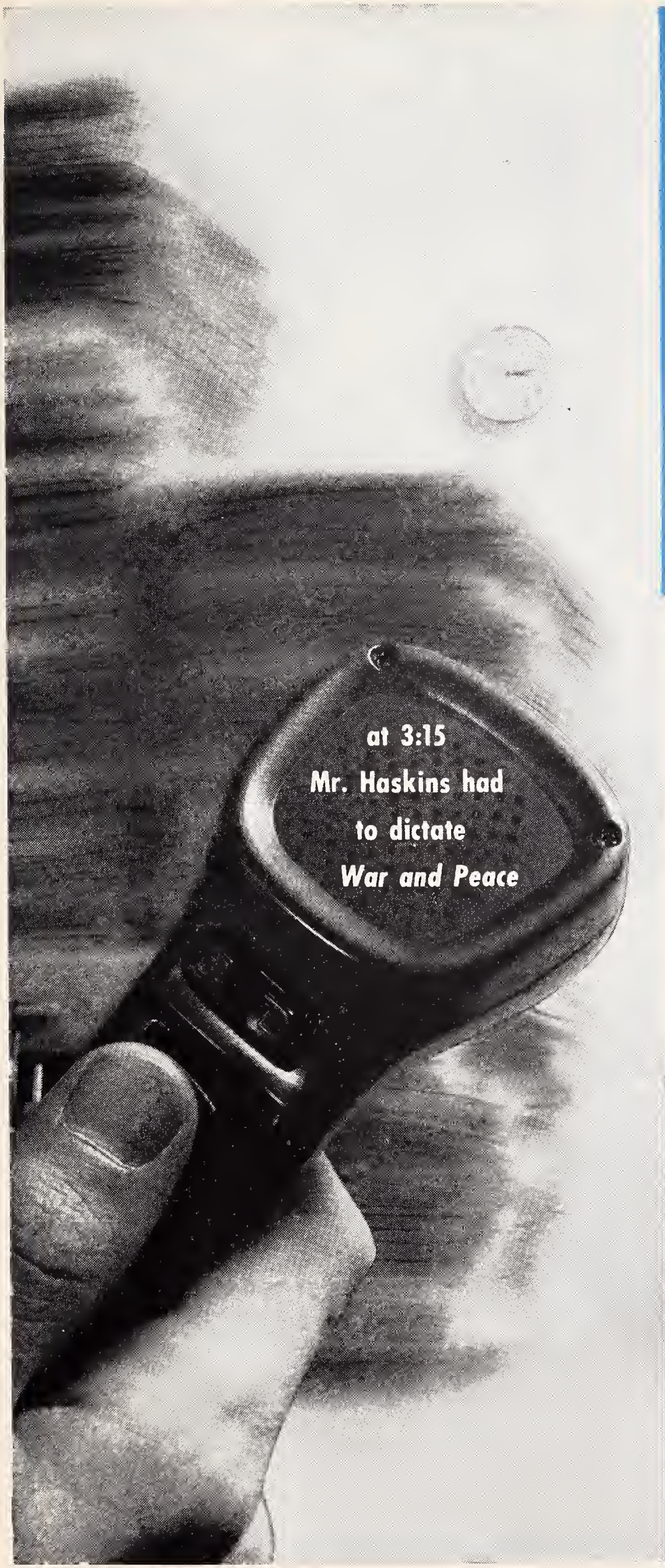

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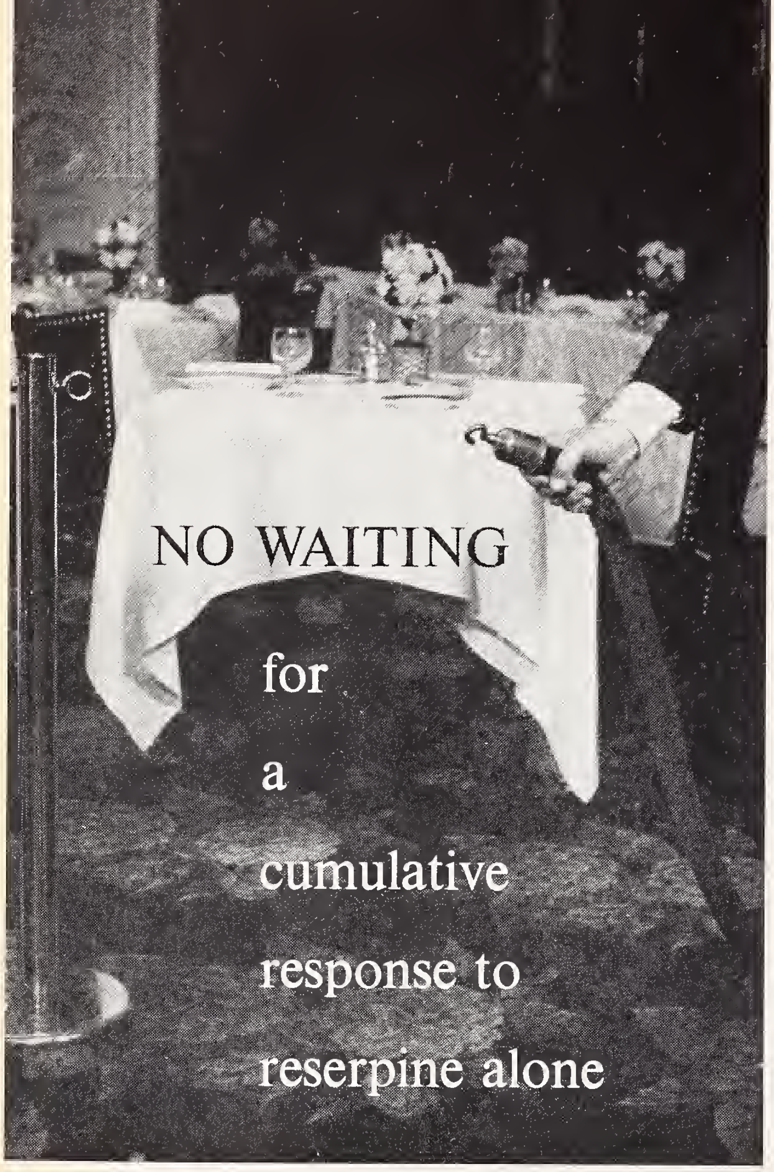

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Woman's Auxiliary

MEDICAL AUXILIARIES OF DISTRICT IV HOLD REGIONAL MEETING

The Auxiliary to the Blue Earth County Medical society held its regional meeting at the Century Club, North Mankato, on Monday, November 25. The auxiliaries of Brown, McLeod, Nicollet-Le Sueur, Scott-Carver, and Waseca are included.

Mrs. C. L. Oppegaard of Crookston, state medical auxiliary president, and Mrs. Reuben Erickson, president-elect, were present at the luncheon. Mrs. Oppegaard informed the group of the coming auxiliary activities.

David Ling, who is in charge of the driver's training program at the Mankato high school, spoke on "The Youthful Driver." He stated that nationally students taking an approved behind-the-wheel, driver-training course had only half as many accidents as their classmates who did not have the course. This figure holds true in Mankato as well. Furthermore, it is the driver who goes out of control, not the car. Hubert Cary, safety supervisor of Northern States Power company, listed rules of safety pertaining to the home, such as stair rails, tacked down carpeting on stairs, care with electrical appliances in bathrooms, and many more valuable tips. It is not the uncommon hazard but the everyday one that causes trouble.

Mrs. Alton Lindblom, of Mankato, District 4 regional advisor to the Minnesota State Medical auxiliary, was in charge of general arrangements. Her committee consisted of Mrs. George Penn, state auditor of the medical auxiliary; Mrs. E. E. Heller, president of the Blue Earth County Medical auxiliary, Mrs. John Heimark, safety chairman; and Mrs. Robert Engstrom.

MRS. W. C. CHAMBERS

Funeral services for Mrs. Anna Chambers, seventy-five, were held November 5, 1957, in the First Presbyterian Church of Blue Earth. She was the wife of Dr. Winslow C. Chambers, Blue Earth physician for more than fifty years.

Mrs. Chambers was a member of the Woman's Auxiliary to the Blue Earth Valley Medical Society. In addition, she was active in many civic and church organizations including units within the Presbyterian church, Faribault County Historical Society, county Republican activities, American Legion Auxiliary, Girl Scouts, Red Cross, Christmas Seals and other health associations and groups. Mrs. Chambers was also the first president of the United Church Women in Blue Earth, one of the trustees of the permanent care fund of the Riverside Cemetery in Blue Earth, and a charter member of the Home Economics Club and the Riverside Town and Country Club.

Surviving are her husband; two sons, Clarke, a professor of history at the University of Minnesota, and Winston, Seattle, Washington; one daughter, Mrs. Sheldon B. Vance, Brussels, Belgium; and six grandchildren, Robert and Stephen Vance, Brussels, Belgium; James Chambers, Seattle, Washington; Jenny, Katherine and Robert Chambers, St. Paul.

General Interest

Dr. J. A. Myers, professor emeritus of medicine and health at the University of Minnesota, has received the American Health Association award in recognition of his leadership in promoting tuberculosis skin testing surveys in schools of Minnesota and other states.

* * *

"Bone Tumors: General Aspects and an Analysis of 2,276 Cases," a new 224-page book by **Dr. David C. Dahlin**, consultant in the section of Surgical Pathology of the Mayo Clinic at St. Mary's Hospital and associate professor of pathology in the Mayo Foundation, has been published by Charles C Thomas of Springfield, Illinois.

* * *

Dr. James W. Kernohan, chairman of the Sections of Pathology of the Mayo Clinic and professor of pathology in the Mayo Foundation, was elected president of the American Board of Pathology at the October meeting of that organization.

* * *

Dr. L. A. Laikola, of Adrian, has been elected president of the Southwestern Minnesota Medical Society. He succeeds **Dr. Victor Doman**, of Lakefield, as president. Other elected officers include: **Dr. F. W. Bofenkamp** of Luverne, president-elect; **Dr. Hugh Patterson** of Slayton, vice president; and **Dr. O. M. Heiberg**, Worthington, secretary.

* * *

Dr. S. A. Slater, Worthington, superintendent, and **Dr. C. L. Sherman**, Luverne, commission president of the Southwestern Minnesota Sanitorium in Worthington, were recently honored for thirty-eight years of activity in the battle against tuberculosis.

* * *

Dr. Richard W. Anderson has resigned his post as chief of the psychiatric service at Minneapolis General Hospital.

* * *

Dr. R. A. MacDonald, Littlefork, will continue his practice in that city, following his resignation as administrator of that community's municipal hospital.

* * *

Dr. Ray C. Anderson, University Hospitals, Minneapolis, has been elected a fellow in the American Academy of Pediatrics.

* * *

Dr. and Mrs. Moses Barron, Minneapolis, were recently honored at the twenty-fifth annual dinner of the

---

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Liver, Desiccated, N.F.	110 mg.
Vitamin B_{12}	1 mcg.
Folic Acid	0.25 mg.
Thiamine Mononitrate	1 mg.
Riboflavin	1 mg.
Pyridoxine Hydrochloride	0.25 mg.
Calcium Pantothenate	0.25 mg.
Nicotinamide	3.3 mg.
Ascorbic Acid	16.66 mg.

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Twin City Chapter, American Friends of the Hebrew University, which was held at the Dyckman Hotel.

* * *

Dr. Thomas William Parkin, Rochester, has been named a fellow of the American College of Physicians. Associates of the College also named include **Dr. Joseph M. Kiely** and **Dr. Raymond D. Pruitt**, both of Rochester; **Dr. Charles Edward Lindemann** of Minneapolis, and **Dr. John K. Meinert** of Willmar.

* * *

Two Minnesotans were among nine medical researchers and physicians who were recently cited for outstanding contributions to medical progress by the 1958 Modern Medicine Distinguished Achievement Awards. They are **Dr. C. Walton Lillehei** and **Dr. Philip S. Hench**, Rochester.

* * *

Dr. Clarence R. Ferrell, Grand Rapids, has been named Itaska County Home physician. He succeeds **Dr. Clifford E. Sisler**, who resigned to accept a position on the health staff of the University of Minnesota.

* * *

Anoka County physicians have named **Dr. Matthew K. Plasha** and **Dr. Harold A. Mork** to serve as a committee to whom any public health matters which concern the community are to be referred in the future.

* * *

Dr. Francis W. Lynch has been elected the 1958 president of the Ramsey County Medical Society. He succeeds **Dr. Allen Wilson.** Other officers elected include: **Dr. Arthur H. Peterson**, vice president; **Dr. Gordon Kamman**, president-elect; and **Dr. Abbott Skinner**, secretary-treasurer.

* * *

Dr. J. T. Anderson, who has practiced medicine at 8488 Central Avenue Northeast, Spring Lake Park, since 1952, has formally discontinued private practice.

* * *

Dr. W. A. Hanson, Minneapolis, has been elected Mississippi Valley Medical Society vice president for the state of Minnesota.

* * *

Dr. Reuben Berman, Minneapolis, has been appointed governor for the state of Minnesota, in the American College of Cardiology.

* * *

Dr. Merrill Chesler, Minneapolis, was the principal speaker at the November meeting of the Third District Minnesota Nurses Association, held at Mount Sinai Hospital. The lecture "Burn Therapy" was given in conjunction with a refresher course for Civil Defense.

* * *

Dr. E. J. Nelson, of Lonsdale, has been elected chief of staff of the Community Memorial Hospital in New Prague. He succeeds **Dr. Elmer Doherty**, of New Prague. **Dr. Olaf Lukk**, of Montgomery, was elected vice chief of staff and **Dr. Elizabeth Rieschl**, of Jordan, was re-elected secretary-treasurer.

Dr. Donald C. Balfour, emeritus director of the Mayo Foundation and former head of a section of surgery in the Mayo Clinic, has been awarded a hand-illuminated certificate which designates him an honorary life member of the Medical Alumni Association of the University of Toronto.

* * *

Dr. Malcolm A. McCannel, has established a temporary branch office at Southdale at 3113 West 66th Street, Minneapolis. This location is in addition to the downtown office, 325 Doctors Building.

* * *

Dr. Edgar V. Allen, internist of the Mayo Clinic and professor of medicine in the Mayo Foundation, Graduate School, University of Minnesota, recently attended a meeting of the Council on Research in High Blood Pressure of the American Heart Association, Cleveland, Ohio. Dr. Allen is the retiring president of the American Heart Association. In Cleveland Dr. Allen also participated in a panel discussion on "The Business Man and His Heart."

* * *

Dr. Gershom J. Thompson, head of the section of urology, has been elected president of the staff of the Mayo Clinic succeeding **Dr. Herman J. Moersch. Dr. Frank H. Krusen**, head of physical medicine and rehabilitation, was elected vice president; **Dr. F. Raymond Keating, Jr.**, first councilor; **Dr. George A. Hallenbeck**, second councilor, and **Dr. Charles A. Owen, Jr.**, was re-elected secretary.

* * *

Dr. Leo G. Rigler has been elected to the presidency of the Radiological Society of North America. Election took place at the 43rd annual meeting of Radiological Society of North America held November 17-22. Dr. Rigler this year began an association with Cedars

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of Lebanon Hospital in Los Angeles as consultant in radiology following a long and distinguished career as director of the department of radiology and professor at the University of Minnesota School of Medicine.

* * *

Three members of the staff of the Mayo Clinic who are also members of the faculty of the Mayo Foundation, and the director of the Mayo Foundation, have been appointed members of the Senate of the University of Minnesota. The four are **Dr. Victor Johnson,** director of the Mayo Foundation and professor of physiology in the Mayo Foundation, Graduate School, University of Minnesota; **Dr. Raymond D. Pruitt,** a consultant in medicine in the Mayo Clinic and professor of medicine in the Mayo Foundation; **Dr. Charles F. Code,** co-chairman of the Section of Physiology in the Mayo Clinic and professor of physiology in the Mayo Foundation, and **Dr. William H. ReMine,** head of a section of general surgery in the Mayo Clinic and instructor in surgery in the Mayo Foundation.

* * *

Dr. John G. Rukavina, specialist in diseases of the skin, cancer of the skin and skin surgery, has opened his medical practice in the Lowry Medical Arts Building in St. Paul. He was formerly Associate Professor of Dermatology at the University of Wisconsin.

* * *

Dr. A. M. Lundholm, formerly of St. Paul, is now associated with the Cambridge Clinic.

Dr. Victor K. Funk has been named assistant superintendent and associate director of Glen Lake Sanatorium. **Dr. Peter M. Mattill,** who formerly held that position, has moved to Dr. Funk's present position as assistant medical director at Glen Lake.

* * *

Dr. Alden F. Wiley, formerly of Kalkaska, Michigan, has joined **Dr. Raymond Chittum** in full-time practice in the East Range Clinic at Hoyt Lakes. Other clinics are at Aurora, Eveleth, and Virginia.

* * *

Dr. Jack V. Wallinga, Minneapolis, has been named psychiatric consultant to the Lutheran Welfare Society of Minnesota.

* * *

Dr. Kermit Halverson, a native of Fergus Falls, has accepted the post of physician at the Chisholm Mesaba Clinic.

* * *

Dr. Charles W. Mayo has been appointed assistant chief medical adviser of the National American Legion.

* * *

Dr. Richard M. Hewitt, emeritus head of the Section of Publications of the Mayo Clinic, Rochester, Minnesota, and assistant editor of the *Journal of the American Medical Association* from 1925 to 1928, has been appointed Alfred P. Sloan Visiting Professor at the Menninger Foundation, Topeka, Kansas, for a six-month tenure. Dr. Hewitt is also emeritus associate professor of medical literature in the Mayo Foundation, Graduate School, University of Minnesota.

* * *

Dr. Harold E. Miller has been re-elected president of the Northwestern Hospital medical staff. Other new medical staff officers are: **Dr. Maynard C. Nelson,** vice president; **Dr. Albert J. Schroeder,** secretary-treasurer (re-elected); **Dr. Harold Buchstein,** chief of surgery; **Dr. Mark C. L. Hanson,** chief of medicine; **Dr. Edgar Ingalls,** chief of obstetrics-gynecology; **Dr. Northrop Beach,** chief of pediatrics, and **Dr. Frederick Hass,** chief of general practice.

* * *

Dr. Donald Honath has been named as chief of staff and president of the medical staff at the Owatonna City Hospital, succeeding **Dr. D. K. Halvorsen** to that post. Named to serve for the coming year with Dr. Honath were **Dr. J. A. McIntyre,** vice president, and **Dr. John Hoff,** secretary-treasurer.

* * *

Dr. Hiram E. Essex, co-chairman of the Section of Physiology of the Mayo Clinic at Rochester, Minnesota, and professor of physiology in the Mayo Foundation, Graduate School, University of Minnesota, delivered the Hatfield Lecture at the meeting of the Philadelphia College of Physicians and Surgeons of Philadelphia on January 8, 1958.

* * *

Dr. Suad A. Niazi, who has been associated with the Hauge Clinic in Clarkfield for the past ten months, has resigned that position to associate himself with **Dr. M. S. Nelson** in the Granite Falls Clinic, effective immediately.

Work has begun on construction of a $90,000 medical clinic at 8 W. Sixty-fifth Street, Minneapolis, for **Drs. C. Gordon Watson, Robert L. Sturgis** and **Henry Brandt.**

* * *

Dr. Jack F. Haas has announced his association with **Dr. S. T. Kucera** and **Dr. G. N. Rysgaard,** in their office at the Medical Arts Building in Northfield.

HYGIENIC GUIDES AVAILABLE

Hydrogen peroxide purchased from the corner drug store in a highly diluted solution has been essential to production of feminine blond coiffures for many years. The same hydrogen peroxide in its concentrated form can be a health hazard to the industrial employe if not properly controlled. Hazards and control of this concentrated chemical in the industrial environment are contained in a Hygienic Guide published in a recent issue of the *Industrial Hygiene Quarterly.* Hygienic Guides on silica, sulfuric acid and dimethylformamide are contained in the same publication. Guides on vanadium pentoxide, xylene, ethylene glycol monomethyl ether, phosphoric acid and toluene were published in a previous issue of the *Quarterly.* A total of forty-four subjects currently comprise the series.

Hygienic Guides are prepared by a committee of the American Industrial Hygiene Association as part of its effort to safeguard the health of the industrial employe. Each Guide covers a single chemical with the latest available information on maximum allowable concentrations for both short and long term exposures, significant chemical and physical properties, major uses, evaluation of exposures and engineering and medical control procedures with literature references. They form a ready reference for industrial hygienists, medical and safety directors, plant engineers and others concerned with the health and safety of industrial employes.

A complete list of currently available Guides can be obtained from the American Industrial Hygiene Association, 14125 Prevost, Detroit 27, Michigan. Single copies of each are also available from the same source at 25 cents each. Remittance must accompany orders of $2.00 or less. Discounts of 20 per cent are allowed on orders for five or more, and 40 per cent for 100 or more. Each Guide is a single sheet, punched for a multiple ring binder. Binders are available at $1.25.

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MINNESOTA BLUE CROSS-BLUE SHIELD

More than $23,490,000 has been paid to hospitals by Minnesota Blue Cross for 1,017,294 days of hospital care incurred by 168,896 participant subscribers during the first ten months of 1957.

The effect of the month of October upon Blue Cross year to date experience was an increase in hospital expense exceeding $2,580,000 representing nearly 109,000 days of hospital care incurred by 17,571 participant subscribers. An increase of 14 per cent was experienced in number of Blue Cross cases paid per year per 1,000 contracts protected during the month of October compared with the preceding month.

Accident cases and conditions related to pregnancy continue to be the leading reasons for the hospitalization of Blue Cross subscribers, however, the number of subscribers hospitalized for respiratory illnesses increased 73 per cent from September to October, 1957.

An increase in frequency of respiratory illnesses incurred is normally anticipated during the last quarter of the year compared with the preceding quarter. This normal increase is a continuing trend upwards to the peak period for respiratory illnesses experienced during the first four or five months of any year. In a review of the number of subscribers hospitalized for respiratory illnesses from July through October, 1957, compared with 1956, it is noted that nearly 1,600 more Blue Cross subscribers incurred hospital care due to respiratory illnesses during this period of 1957 than during the same period of the preceding year—thirteen more cases per day admitted—or one every two hours.

Respiratory illnesses accounted for 14.3 per cent of the total cases paid during the month of October, 1957, and represented 72 cases per year per 1,000 contracts protected compared with 11.9 per cent representing 58 cases paid per year per 1,000 contracts protected during October, 1956.

Minnesota Blue Shield paid physicians $7,113,817 during the first ten months of 1957 for medical-surgical-obstetrical and related services rendered participant subscribers. This figure exceeds by more than $1,400,000 or over 24 per cent, the amount paid physicians for care of participant subscribers during the first ten months of 1956. From January 1, 1956, through October, 1956, Blue Shield paid $5,707,041 in the form of subscriber benefits.

Blue Shield payments to physicians during the first ten months of 1957 exceeded, by more than $190,000, the amount paid during the entire year 1956 for doctors' services rendered participant subscribers. Blue Shield provided subscriber benefits of $6,919,348 during the twelve months of 1956.

During the first ten months of 1957, Blue Shield provided benefits for 258,728 physicians' services rendered participant subscribers. This figure is more than 24 per cent greater than the number of physicians' services, 206,245, for which Blue Shield provided benefits during the same period in 1956.

Participant subscribers numbered 859,327 as of October 31, 1957, or 55,512 more than the number of participant subscribers enrolled as of the same date in 1956. Minnesota Blue Shield participant subscribers numbered 803,815 as of October 31, 1956.

Vaginal carcinoma is uncommon and usually starts on the posterior wall in the upper third of the vagina.

* * *

Sarcomas of the female genital tract usually arise in the body of the uterus.

* * *

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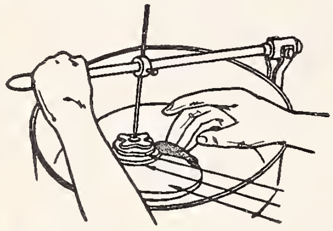

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Book Reviews

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

THE INCURABLE WOUND. By Berton Roueche. 177 pages. Price $3.50, cloth. Boston: Little, Brown & Company, 1958.

MODERN SEX LIFE. Edwin W. Hirsch, M.D. (A Signet Book). 160 pages. Price 35c, paper cover. New York: The New American Library of World Literature, Inc., 1957.

SURGERY IN WORLD WAR II. OPHTHALMOLOGY AND OTOLARYNGOLOGY. Editor in Chief, Colonel John Boyd Coates, Jr., MC. 605 pages. Illus. Price $5.00, cloth. Washington, D. C.: U. S. Government Printing Office, 1957.

BONE TUMORS. General Aspects and an Analysis of 2,276 Cases. David C. Dahlin, M.D., Consultant, Section of Surgical Pathology, Mayo Clinic; Associate Professor of Pathology, Mayo Foundation, Rochester, Minnesota. 224 Pages. Illus. Price, $11.50, cloth. Springfield, Illinois: Charles C Thomas, 1957.

DISEASES OF THE NOSE, THROAT AND EAR. Howard Charles Ballinger, M.D., F.A.C.S., Professor Emeritus, Department of Otolaryngology, Northwestern University Medical School, Chicago; Surgeon, Department of Otolaryngology, Evanston Hospital, Evanston, Illinois. John Jacob Ballinger, B.S., M.D., Associate, Department of Otolaryngology, Northwestern University Medical School, Chicago; Associate Surgeon, Department of Otolaryngology, Evanston Hospital, Evanston, Illinois. 10th edition. Philadelphia: Lea and Febiger, 1957.

This tenth edition of a well-known and very popular textbook on diseases of the ear, nose and throat manifests many improvements over the previous editions. Many subjects are brought more up to date; e.g., physiology of the nose, eustachian tube and hearing; physiology and function of the labyrinth; malignant diseases of the nose, throat and ear; allergy and its manifestations; Ménière's disease; endoscopic diseases and their management, et cetera. This text is a complete discussion of diseases of the ear, nose and throat. Each subdivision covers the anatomy, treatment and complications of diseases of that part. This has been characteristic of previous editions and is an excellent help in teaching. All in all, this is an excellent book and in my opinion, will fill the need for a text and reference work in otolaryngology as was hoped by the authors. It will serve not only the student but also the practicing otolaryngologist as well.

Some of the sections are particularly outstanding. The chapter describing the physiology of the nose and sinuses with their treatments includes the most up-to-date information and is well written. The chapter on allergy is concise and covers the essential information without a lot of unnecessary material. The chapter on malignant growths of the face, nose, mouth, pharynx, larynx and trachea is concise and well illustrated. It covers with minimum information the problems most

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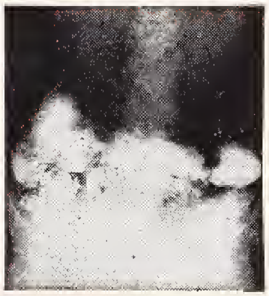
when anxiety and tension "erupts" in the G. I. tract...

IN ILEITIS



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The Minnesota State Medical Association maintains a Medical Placement Service for the benefit of physicians who are looking for locations and positions; also for communities, medical groups and physicians who are looking for licensed medical assistance. For information, write to the Minnesota State Medical Association, 496 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota.

(Continued from Page A-53)

commonly encountered in otolaryngology and leaves out much of the more detailed information which is best left to this one subject alone.

Another attribute about this text to me is its discussion of many of the smaller fields in this specialty which are so necessary to successful treatment of ear, nose and throat diseases; e.g., treatment of epistaxis, headache, neuroses of the larynx, defects of speech, impacted cerumen, deaf mutism and aural rehabilitation. However, these small sections could well be expanded. The chapters on endoscopy by Gabriel Tuker, Chevalier Jackson and Joseph Atkins are a valuable part of this text as they cover a vital part of the modern otolaryngologist's practice.

The criticisms of the text are but few and will undoubtedly be corrected in future editions. Most of the criticisms stem from the rapid recent advances in otolaryngology which renders a text almost obsolete before it can be published.

As mentioned in the preface, this book has evolved during its various editions into somewhat of an encyclopedic type not due to the original intention of the authors, but by the gradual inclusion of many of the less well-known clinical entities during the almost fifty years of its existence (first edition, 1908). The authors would do well to eliminate much of this encyclopedic, merely historical information from a text which is intended to serve the busy otolaryngologist and the student whose time is already too well filled with essential information; for example, the elimination of obsolete tests for hearing which are only of historical interest, operations which have been considered obsolete by modern otolaryngologists, and the description of equipment which in the eyes of present surgeons is no longer of much use. A great deal can be accomplished to make this book more concise and more helpful by such eliminations.

The material on otology could be improved. The impression is left that the surgical procedures described many years ago for the treatment of ear disease are still as well accepted as endaural procedures which are now almost universally employed in the large teaching institutions. As a matter of fact, as much space is given to the post auricular approach to the middle ear using hammer and chisel as is given to the modern endaural procedures. Stapes mobilization, tympanoplasty, selection and fitting of hearing aids, the management of deafened patients receive inadequate discussion.

As a practicing otolaryngologist, I believe that the difficulties most commonly seen in practice should be given more discussion; for example, allergy of the ear, nose and throat, pains in various locations of the head and neck, tumors and their treatment, endoscopic problems, fitting of hearing aids, management of the deafened patient.

William F. Andrew, M.D.

---

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REFERENCES (1) Woolington, S. S.; Adler, S. J., & Bower, A. G., in Welch, H., & Marti-Ibanez, F.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 365. (2) Ditmore, D. C., & Lind, H. E.: *Am. J. Gastroenterol.* 28:378, 1957. (3) Hasenclever, H. F.: *J. Iowa M. Soc.* 47:136, 1957. (4) Waisbren, B. A., & Strelitzer, C. L.: *Arch. Int. Med.* 99:744, 1957. (5) Holloway, W. J., & Scott, E. G.: *Delaware M. J.* 29:159, 1957. (6) Rhoads, P. S.: *Postgrad. Med.* 21:563, 1957. (7) Petersdorf, R. G.; Bennett, I. L., Jr., & Rose, M. C.: *Bull. Johns Hopkins Hosp.* 100:1, 1957. (8) Royer, A.: Changes in Resistance to Various Antibiotics of Staphylococci and Other Microbes, paper presented at Fifth Ann. Symp. on Antibiotics, Washington, D. C., Oct. 2-4, 1957. (9) Doniger, D. E., & Parenteau, Sr. C. M.: *J. Maine M. A.* 48:120, 1957. (10) Josephson, J. E., & Butler, R. W.: *Canad. M. A. J.* 77:567 (Sept. 15) 1957.

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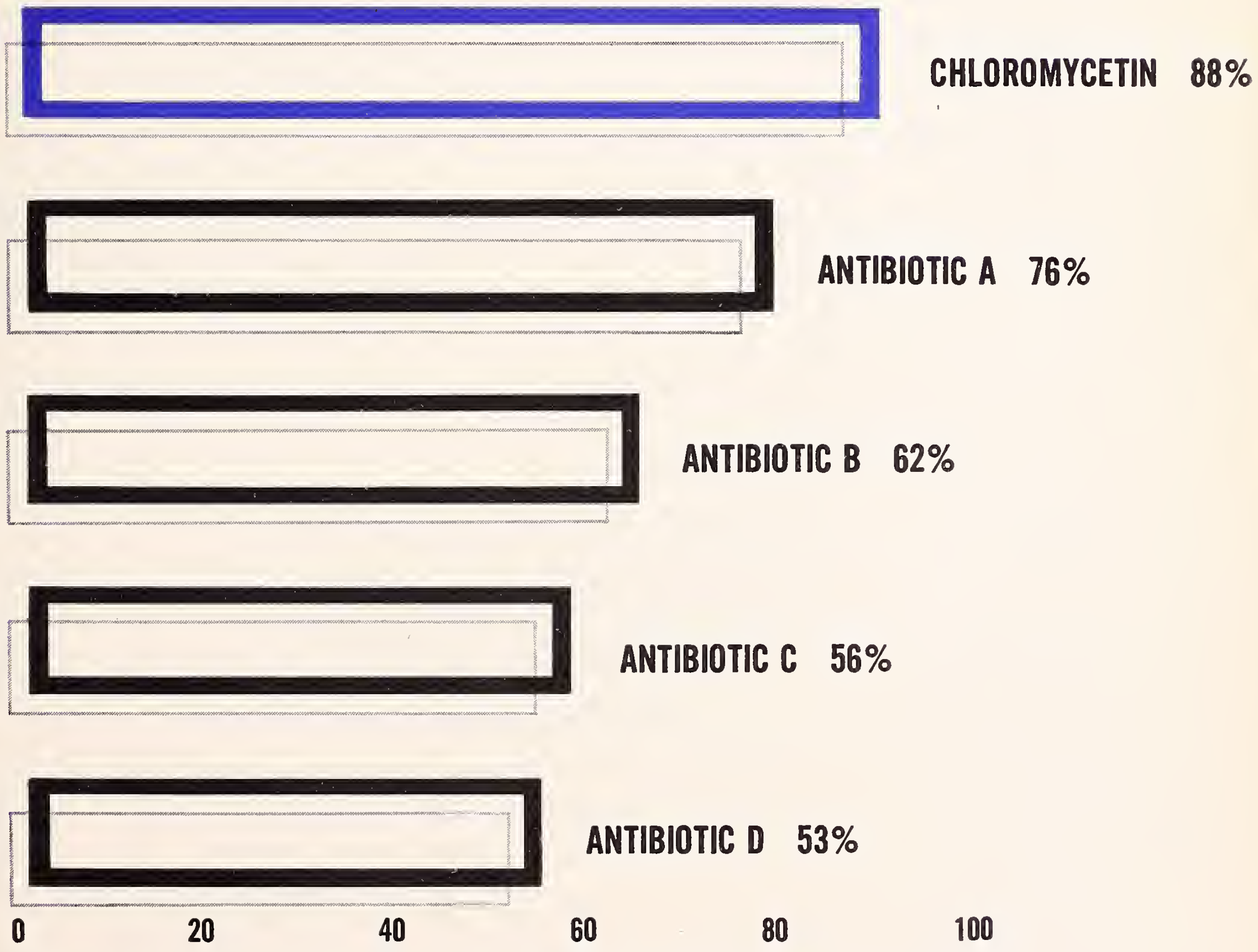

*Adapted from Ditmore and Lind.[2] Organisms tested were isolated from stools of 48 patients.

25058

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there's pain and
inflammation here...
it could be mild
or severe, acute or
chronic, primary or
secondary fibrositis — or even
early rheumatoid arthritis

Original Contributions

Acute Diarrhea of Infants and Small Children

A Review

THOMAS E. REICHELDERFER, M.D., M.P.H.
Bethesda, Maryland

DIARRHEA is not in itself a disease but rather a symptom of illness. It is characterized by frequent evacuation of the bowels with unformed stools, accompanied by large amounts of water, body electrolytes, and undigested food. It is well for this reason to discuss diarrhea as a single entity even though the etiology may vary from any of several bacterial agents, viruses, parasites, foods and emotional crises.

The treatment of diarrhea is directed primarily at the correction of the physiological disturbances produced by loss of water and ions, and secondly against the causative agent. Infants, because of the larger proportion of water making up the body mass, are more prone to suffer from severe dehydration and electrolyte loss than adults or older children. Three hundred to 500 cc. of water may be lost in the stools. Recent investigations have uncovered agents that are responsible for epidemic diarrhea of the newborn and parenteral diarrhea.[1]

The determination as to whether or not a child has diarrhea is usually a simple one. The parents report the use of an increased number of diapers soiled with a loose stool, accompanied by a large water ring. The child often asks to be placed on the pot but will frequently have an evacuation before the parent can undress him. The character of the stool in small infants is important. It is usually green, contains mucus and in the severe cases may be streaked with blood or be grossly bloody. The frequency is increased over the normal pattern for bowel movements. The transitional stool of the newborn with its green color often confuses young mothers sent home too early from the hospital. The lack of frequency and mucus and the change to yellow stools in a short time are often enough to reassure the mother and exclude diarrhea.

Since Colonial times, the summer with its high incidence of diarrhea (cholera infantum) has always been a particularly hazardous period for infants and small children. The author's grandmother, in the years following the Civil War, gave birth to thirteen children. Only six reached their majority. The remaining succumbed shortly after birth or during the first year of life to "summer complaint" or infantile diarrhea. Conditions changed little until about 1900. Diarrhea was at that time among the leading causes of death in the first year of life. The rates for deaths due to diarrhea and enteritis have fallen with the passage of time in those states which require registration. In 1900 the rate was 142.7 deaths per 100,000 population; 1920, 53.7; 1930, 26; 1940, 10.3 and 1950, 4.9.[2]

The production of pasteurized and evaporated milks, refrigeration, pure water supplies and improved methods of sewage disposal have accounted in part for this reduction in infant mortality. The introduction of chemotherapeutic and antibiotic drugs and widespread use of parenteral fluids have brought about a further reduction in mortality due to diarrheal disease.

The known agents that cause gastroenteritis have changed little with the passage of time. They will always remain as a threat to our population. Some have become so rare that physicians rarely see a case caused by them. New causative agents are being discovered. The initial treatment in each is directed at the correction of physiological disturbances produced by the diarrhea. The following is a short description of some of the diarrhea-producing organisms, substances and states accompanied by a brief commentary.

Presented at the annual meeting of the Northern Minnesota Medical Society, Alexandria, Minnesota, September 7, 1956.

Dr. Reichelderfer, formerly Assistant Professor of Pediatrics, University of Minnesota, is now at the Laboratory of Infectious Diseases, National Institutes of Health, Bethesda, Maryland.

Parasites

There are many parasitic agents responsible for diarrhea. The most frequently encountered in children are *Endoamoebia histolytica, Strongyloides, sterocoralis, Giardia lambia* and *Balantidium coli.* All are spread by fecal contamination. These infections are not common in the latitude of Minnesota. They are discussed only for completeness and with the thought that we are living in a world of ever shrinking distances.

The acute phase amoebiasis (*E. histolytica*) requires attention to water loss as in every other diarrhea. Treatment with Terramycin® (oxytetracycline) will destroy large numbers of amoebia and cysts in the colon. Most authorities agree that the broad spectrum antibiotics should be administered with chloroquine and Diodiquin.® Amoebiasis in this part of the world is usually spread by faulty plumbing connections and contaminated water and food.

Strongyloides sterocoralis is found in the soil mostly in warm climates. The worm burrows in the skin of the bare feet, is carried to the lungs and thence coughed up and swallowed into the gastrointestinal tract. There the tiny worms burrow into the crypts of the mucosa of the colon producing necrosis of tissue and a severe diarrhea. Treatment is generally unsatisfactory and the disease becomes chronic. Enteric coated tablets of gentian violet have been used with some success.

Giardia lambia is a flagellate that has been implicated in epidemics of diarrhea in children[3] and young adults. Several of the medical students at the University of Oregon in 1955 were found to have gastroenteritis from this organism. Treatment with atabrine appears to be most efficacious.

Balantidium coli is a ciliate which is usually found in the gastrointestinal tract of swine. Human infections are accidental. Severe diarrhea is produced by this organism. Fortunately, oxytetracycline effectively destroys it.

Fungi

Candida albicans has been implicated in several epidemics of diarrhea of the newborn.[4,5] Diarrhea following widespread use of broad-spectrum antibiotics (allowing C. albicans to replace the normal flora of the gut) has become common in recent years. Infants may be treated wtih gentian violet. Mycostatin® is specific for this organism and should be used whenever possible. Other fungi when ingested with spoiled foods may cause a transitory gastroenteritis.

Bacteria

Staphylococci.—Staphylococcal food poisoning with its resulting gastroenteritis is the result of food being contaminated by a nasal or skin carrier or by open staphylococcal lesions. Frequently the food is allowed to stand several hours before serving during which time the potent enterotoxin is formed. Ham, cream puffs, eclairs and salads frequently are incriminated. Several epidemics have been shown to have their origin in community or church suppers. The onset of the gastroenteritis is sudden, usually within one-half to four hours. Treatment is supportative during the acute phase with emphasis on the replacement of fluids lost in the crampy, frequent stools that are passed. Pasteurization of bakery products, refrigerated storage and elimination of infected food handlers are effective means of prevention.

Severe pseudomembranous enterocolitis from resistant strains of staphylococci may appear as a complication in those patients who have been treated with antibiotics,[6] the staphylococci having replaced the natural flora of the intestines.

A recent epidemic of diarrhea in the newborn nursery of a St. Paul Hospital was shown to be due to Staphylococcus aureus.[7] All affected infants had this organism in their stools and nasopharynx. Several of the nurses harbored this same organism in their nose and throat.

Enteric Bacilli.—The enteric bacteria causing diarrhea in infants and children are those of the following genera: *Vibrio, Shigella, Salmonella, Proteus, Pseudomonas, Paracolon* and *Escherichia.* All are characterized by their inability to ferment lactose in culture media with the exception of the *Escherichia.* The most important members of this group of enteric pathogens in this part of the world are the *Salmonella, Shigella* and the enteropathic coliforms of the genus *Escherichia.* Generally, the drugs that are most effective against this group of organism (in order of their activity)[8] are: Chloromycetin® (chloramphenicol), broad spectrum drugs of the mycin group, sulfadiazine, and neomycin.

Laboratory techniques for the isolation of these organisms have been improved greatly. The use of selective media such as selinite, desoxycholate and desoxycholate-citrate permits rapid differentiation between pathogens and nonpathogens in the stools. Colonies may be picked from these selective media and placed in sugars for accurate identification. The development of specific sera for agglutination of the enteropathic coliforms and a polyvalent sera for the *Salmonella* and *Shigella* has hastened laboratory diagnosis.

Shigella usually reach the gastrointestinal tract by the media of infected food and water.[2,9,10] The incubation period varies from one to seven days. The pathological process is an inflammatory one which affects the colon and often the terminal ileum. Abdominal cramps and discomfort are present. Blood and mucus are found in the stools. A smear of the mucus, when stained, will show a predominance of polymorphonuclear leukocytes. This will often help to separate a *Shigella* dysentery from a *Salmonella* dysentery before laboratory reports are available. Infants occasionally show meningismus, jitteriness and convulsions.[11] The neurotoxin produced by the *Shigella* produces this effect. The diagnosis of dysentery is often made at the time of a diagnostic lumbar puncture when a foul, loose, bloody, mucous-containing stool is passed as a result of the trauma involved in the procedure. Sulfadiazine and other less soluble sulfa drugs are used to treat shigellosis. Chloromycetin and other mycin drugs may also be used. Polymyxin is effective in clearing up the carrier state. Epidemic diarrhea in nurseries due to this organism is rare.

Salmonella.—The bacteria of the *Salmonella* group include over 150 different strains, the majority of which are animal pathogens.[12] Clinical symptoms may be those of a mild gastroenteritis but more frequently they are typical of an explosive type of food poisoning. Less frequently, the clinical picture is that of a septicemia, or a typhoidal illness.

The old designation of the typhoid organism *Eberthella typhosa* has been changed. It is now grouped with the *Salmonella* and called *S. typhosa.* Typhoid fever is a classic enteric fever. The fever may be accompanied by either diarrhea or constipation after a seven to fourteen-day incubation period. The patient has a generalized septicemia. The organism multiplies in the lymphoid tissue of the small intestines and regional lymph nodes. Intestinal perforation is often a fatal complication. The biliary tract and bone marrow are often infected—thus a chronic carrier state and osteomyelitis may result. Relapses are frequent (10 per cent). Chloromycetin® is the specific antibiotic for the treatment of this condition. Some authorities have used, in addition, ACTH and the other adrenal steroids. The mortality has been reduced by supportative theory and chloromycin.

Enteric fevers caused by *S. paratyphi* A, B and C and *S. shotmulleri* are of shorter duration and often milder than *S. typhosa.* The treatment is the same.

Gastroenteritis produced by the other members of the group of *Salmonella* have a shorter incubation period, eight to forty-eight hours. The patient usually has diarrhea with an accompanying septicemia. The effects of the disease are milder than with either typhoid or paratyphoid fevers. The antibiotic that is most effective in these infections is Chloromycetin. Chronic carriers and subclinical attacks account for the spread of these conditions in humans. *Salmonella* of this group have the species name frequently designating the place where the strain was first isolated, (*S. Montevideo, S. Newport, S. Panama,* etc.) or the name of the animal in which they are most frequently found, (*S. cholerae suis, S. typhimurium, S. gallinarum*).

Contaminated water, shell fish, milk, and community gatherings have been largely responsible for typhoid fever epidemics. Contaminated foods and powdered eggs[13] have accounted for the milder types of salmonellosis. Hardy recently isolated large numbers of *Salmonella* from processed meats of a Florida abattoir.[14]

Pseudomonas and Proteus.—*Proteus* and *Pseudomonas aeruginosa* are enteric bacilli frequently encountered in normal stools. Their sole appearance in diarrheal stools of infants has led many pediatricians to consider them as causative agents of disease.[15] *Proteus morgani* and *P. mirabilis* seem to be found in sick children more frequently than *P. vulgaris.* Replacement of the usual flora of the colon by these antibiotic-resistant organisms has provided a perplexing problem. Occasional strains are sensitive to Chloromycetin and sulfadiazine but most are resistant to all antibiotics. Oral polymyxin and neomycin are often efficacious. Administration by the oral route avoids the nephrotoxic effect of these drugs since they are not absorbed.

Vibrio comma, the causative agent of Asiatic cholera, is not found in the United States. Replacement therapy and, to a lesser extent, the antibiotics have robbed this disease of much of its past terror.

Enteropathic Coliforms.—Epidemic diarrhea of the newborn was long thought to be due to a viral agent because no pathogenic organisms could be recovered from the stools. Adam[16] in 1927 found certain fermentative types of *E. coli* associated with diarrhea in infants in Germany. Kauffman[17,18] of Denmark listed an antigenic schema for the *E. coli* group in 1944 and 1947. Bray[19] in 1945 in England using Kauffman's antigenic schema associated a specific type of *E. coli* (O 111:B4) with an epidemic of diarrhea among newborn infants. Findings by several workers[20,22] have shown further that certain serologic strains of *E. coli* could cause gastroenteritis. These bacilli had been overlooked when routine methods of screening stools were used.

Classification is based upon identification of *E. coli* serotypes. Special typing sera is required for their identification. Commercial sera have recently appeared on the open market for this purpose. They are simple to use and can be employed in most laboratories. The 0 antigen or somatic structural antigen is thermostable while the B or K antigen from the envelope or capsule is thermolabile. Identification is based on these properties. There have been twelve serological types and subtypes incriminated[23]: O 111:B4, O 55:B5, O 26:B6, 112a, 1126:B13, O 112a, 112c:B11, O 86:B7, O 119:B14, O 125:B15, O 126:B16 and O 127:B8. Other types will probably be implicated in the future. These enteropathic coliforms were found when several epidemics of diarrhea of the newborn were investigated from stool specimens that had been frozen and stored.[24] Stock and co-workers[25] in the Pittsburgh Children's Hospital showed how an epidemic of twenty-five cases in that hospital spread to five other hospitals and nursing homes in the community and accounted for fifty-one secondary cases.

When epidemic diarrhea is found in a nursery these bacilli and other pathogens must be searched for diligently. The exposed infants should be isolated and observed for at least two weeks. The routines of the nursery should be examined for breakdown of isolation techniques employed by the nurses, formula processing, et cetera. Concurrent disinfection of all discharges and all soiled articles must be undertaken. Terminal sterilization of the equipment and nursery is also required. No new admission should be allowed to the nursery. Known cases should be isolated and treated with neomycin 50 mgm/kilogram body weight and given fluid replacement therapy. The high mortality associated with this disease requires that rigid measures be instituted promptly.

Harris et al[26] isolated enteropathic coliforms from five of thirty-five epidemics of diarrhea of the newborn in New York State in 1946-55. It was of interest that the hospitals with the larger maternity services were involved more frequently than the smaller ones. The case fatality rate for diarrhea from these enteropathic coliforms was 28 per cent.

Viral Agents.—The local physician with his "virus X" and "virus disease" was well aware of the viral nature of diarrhea long before the sophisticated researcher was able to isolate the viruses!

Viral agents as causes of diarrhea have only recently been proved to be important. Diarrhea was known to accompany poliomyelitis and some of the other acute viral infections, but studies in epidemic diarrhea of the newborn for viral agents had been disappointing. Buddingh and Dodd[27] isolated (and were able to keep growing in the cornea of rabbits) a virus that they believed to cause diarrhea in infants. Light and Hodes[28] were able to pass another virus (isolated from an epidemic of diarrhea in newborns) using calves. They showed that the calves had a rise in antibody titre to this agent. Not until Enders and his colleagues[29] developed the methods for isolating virus in tissue culture did the laboratory worker have a tool that enabled him to readily isolate viruses. A few viruses could be grown in chick embryos but this was a limited technique. A large number of new viruses have been isolated from the stools of sick and well children during the past two years by the tissue culture technique of Enders. The exact relationship of many of these viruses to disease is not known.[30,32] Sabin[33] at the Cincinnati Children' Hopital examined the tools of fifty-six children, less than four years of age admitted for gastroenteritis, for pathogens and virus. He was able to recover a viral agent in 43 per cent of the cases. Bacterial pathogens in the seventeen patients from whom viruses were isolated included two *Shigella*, one *Salmonella*, one *Paracolon* and three *Proteus*. Viral isolations from monkey kidney tissue culture gave three poliomyelitis type 1 viruses, three adenovirus, three Coxsackie, 2 ECHO viruses (Enteric cytopathic human orphan virus) and nine unknown strains. Forty per cent of the children in a Swedish epidemic of Type 3 adenovirus were reported to have had gastroenteritis. It has long been known that the common cold often manifested itself with grippe-like symptoms and gastroenteritis. Many doctors called this intestinal flu. In light of recent discoveries these were probably adenovirus or ECHO viruses.

Other Agents Causing Diarrhea.—Infants that are overfed and receive either too much carbohydrate or protein in the diet will develop diarrhea.[34] Ingested poisons and cathartics will also cause loose, watery stools. These causes can be uncovered by taking a careful history when the child is seen.

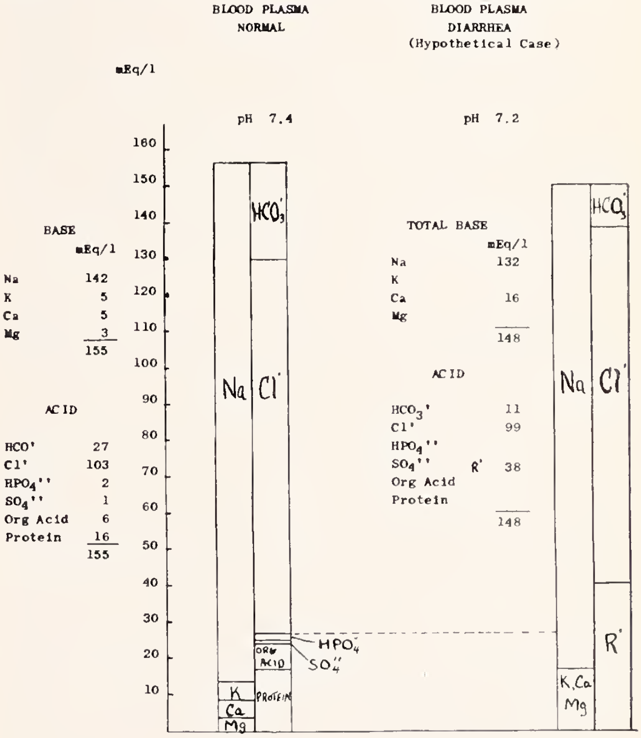
Diarrhea that accompanies pellegra is rarely seen in these days of prosperity and improved diets. Nicotinic acid supplementation of refined foods and more-varied diets have practically eliminated this vitamin deficiency state. Tropical sprue with its diarrheal stools has responded to the administration of folic acid.

Coeliac disease and cystic fibrosis of the pancreas (mucovisidosis) often produce loose, watery stools of a chronic nature. The clinician seeing an infant for the first time should keep these conditions in mind. Especially, when after a trial of adequate treatment, the diarrhea continues and no pathogenic agent is found.

Older doctors often attributed diarrhea to infections elsewhere in the body. These were called parenteral diarrhea, alimentary intoxication or acute nutritional disturbance. Infants often had a myringotomy to relieve their diarrhea.[35] Doubtless, some of these infections did cause loose stools. However, with the use of tissue culture techniques for viral isolation and the discovery of the enteropathic coliforms, many of these uncertain diagnoses will be reported less frequently.

Who cannot recall the diarrhea that accompanies severe states of emotional tension, particularly at examination times. Finally, and not to be forgotten, is the diarrhea that follows the ingestion of certain foods[36]—particularly, that which followed a glorious adventure into a melon patch or orchard where unripe and forbidden fruits were eaten.

Although the causes of diarrhea are varied, the physician, after once establishing that a child has diarrhea, takes steps to identify the organism or to form a hypothesis as to the most likely causative agent. The type of fluid replacement therapy required depends upon the clinical evaluation of the patient—thus treatment is directed towards the patient as well as the cause of his disease.



After Gamble

Fig. 1. Comparison of blood plasma determinations between normal and a hypothetical case of diarrhea.

Symptoms

Clinical observations are as important as the result of laboratory studies in the assessment of diarrhea. In infants there is loss of weight, the skin loses its elasticity and may be pulled up in folds, the eyes and fontanelles are sunken and oliguria may be present. These are all signs of depletion of interstitial fluid and plasma volume. As the diarrhea progresses, circulatory insufficiency develops, the peripheral pulses become feeble, the extremities become cold and ashen and the rate of the heart is increased. The infant is restless and irritable. The child, as circulatory collapse progresses, becomes comatose and may appear cyanotic or pallid. It is remarkable to see these signs disappear when adequate fluid replacement therapy is given.

The diseases causing diarrhea contribute impairment of absorption of water and solutes from the gut, and the subsequent loss of additional water and electrolyte in the stools. The ions lost in the stool include sodium and chloride from the extracellular fluid and potassium, magnesium, calcium and phosphorus from the cells and bone. Infants in the early stages of diarrhea often maintain their nutritive, water and electrolyte balance as long as they continue to eat and drink. Refusal to feed and supervening vomiting occur as the diarrhea progresses. The children are then unable to keep up and replace the loss of gastrointestinal secretions lost in the stools. The additional effects of infection and fever cause depletion of liver glycogen and breakdown of cell protein. Loss of interstitial fluid and plasma volume causes further impairment of the circulation, diminution of renal flow and oliguria. These changes are represented after the manner of Gamble in Fig. 1.[37] The electrolyte and water regulating mechanism of the kidney is lost with the retention of the products of cell catabolism. Alteration in the composition of the body fluids may be reflected in the renal tubules, central nervous system and cardiac muscle. The usual finding in diarrhea is that of a metabolic acidosis due to the greater loss of sodium than chlorides in the stool and the reduction in plasma volume. Water loss may be disproportionate to electrolyte loss and a high serum sodium may be seen in some cases. Alkalosis is rare in diarrhea but has been reported by two observers.[38,39]

Treatment

The method of treatment for diarrhea varies from one physician to another. All, generally follow the comprehensive and systematic plans of Park,[40] Powers,[41] Darrow,[42] Hartman, [35] Butler and Talbot,[43] Gamble,[44] Dodd and Rapoport[45] and Harrison.[46] The emphasis is upon, "the administration of fluids, the transfusion of blood, the withholding of food for a period of time and the administration of food at the end of starvation in gradually increasing amounts."[41] Present-day techniques of fluid administration and electrolyte replacement have greatly simplified therapy. Harrison's method[46] of treatment, because of its ease of adaptibility and simplicity, is recommended for the practicing doctor.

The infant with mild diarrhea from infection may require only simple management at home. Antibiotics, withholding food, and the feeding of an oral solution made of one and a half tablespoons of sugar and half teaspoon of salt to one quart of water may be all that is required. The more colorable physician may prefer to use a commercially-available solution (Lytren) for the oral solution to replace electrolyte. The withholding of solid foods lessen the abdominal distention and decreases the volume of the diarrheal stools. Feedings are gradually resumed over a period of several days. Usually the infant may be put back to breast or started on skim milk feedings when the number of stools has decreased. Over a period of four to five days, the infant is gradually restored to his full caloric requirements.

Chung[47,48] has questioned the advantage of withholding food. He showed in a small series of patients with diarrhea that the intestinal absorption of nitrogen, fat, sodium, calcium, potassium and chloride was improved on a high dietary intake even though the amount of loss in the stools was increased. He compared the clinical courses of infants with diarrhea on "starvation" and "full feeding" regimes and, obtained a more favorable response in those infants on the full-feeding regime. Most clinicians still adhere to Power's method of withholding food.

Opiates and bismuth preparations have little benefit in children on the course of loose stools. Pectin and kaolin may thicken the stool for asthetic purposes. The use of cathartics only further irritates the bowel and aggravates water loss. It is well to keep in mind that the fluid and electrolyte replacement therapy is the most important part of treatment and that little is gained ~~by~~ these additional measures.

The generalist treating mild diarrhea can undertake replacement therapy with meager clinical information. Severe diarrhea that is accompanied by anorexia, vomiting, severe dehydration, circulatory collapse, renal failure, and shock requires, in addition to the data obtained by physical examination and history, the support of a clinical laboratory. An infant with severe diarrhea should be hospitalized. The following laboratory determinations should be performed on admission: Complete blood count (including hemoglobin and hematocrit), CO_2, chloride, sodium, potassium, pH and NPN determinations of the blood. The physician, smaller hospital or clinic—unable to afford the luxury of a clinical laboratory—may resort to Scribner's[49,50] methods for bedside deter-

ORAL ELECTROLYTE SOLUTION (LYTREN)*

Composition	mEq/L			
KC1, NaC1, Na Citrate	Na	50	Citrate	30
NaH_2PO_4, Ca Lactate	K	20	SO_4	4
$MgSO_4$ and Dextri-Maltose	Ca	4	Lactate	4
	Mg	4	PO_4	10
			Chloride	30

10 gm. to 120 cc. water
Mead Johnson and Company

minations of CO_2 and chlorides. *The main value of chemical determinations is to serve as a support of clinical judgment.* Accurate records of fluid administration should be kept day by day, and checks made of the serum electrolytes to support the clinical status of the patient. The simple measurement of body weight supplies an excellent index of the degree of rehydration.

The infant suffering from the effects of severe dehydration and electrolyte loss should receive immediate emergency treatment by parenteral routes. Intravenous infusions of 10 per cent glucose, 20 cc/kilogram body weight and plasma or whole blood in the same amount, are used. These increase circulating blood volume, interstitial fluids, provide water so that circulation, renal blood flow and urine output will be improved, and replace liver glycogen. This is followed by an infusion of a mixture of Lactate-Ringer's solution† and 5 per cent glucose solution in equal parts—80 cc/kilogram body weight. Slow subcutaneous infusion at the rate of 0.5-1.0 cc/min is satisfactory for most infants. This phase of treatment is usually accomplished during the first twelve hours.

Infants may be considered improved if skin turgor is more normal, they are more reactive and alert, voiding frequently and have good peripheral circulation. They are usually thirsty, and oral feedings of electrolyte and carbohydrate solutions (Lytren)** may be begun despite the continual stooling. The oral electrolyte solutions are given six to eight times a day in the amount of 150 cc. or more per kilogram body weight. This solution contains sodium, potassium and chloride. There is no need for potassium replacement in the acute phase of the diarrhea. This solution will provide 3 mEq of potassium per kilogram of body weight per day. This is sufficient for replacement even though some is lost through the stools. Oral feedings of electrolyte solutions are usually continued for two to three days and milk feedings gradually introduced until maintenance calories are provided. Infants that are anemic and malnourished may require transfusions and a high-caloric balanced diet after they have responded to the initial phase of treatment.

When the etiologic agent of the diarrhea is known, the appropriate antibiotic is given with the replacement therapy. When it is unknown, Chloromycin and sulfadiazine or the other broad-spectrum antibiotics, seem to be the drugs of choice. Vitamin C and the soluble vitamins of the B-complex may be given parenterally in severely ill children.

RINGER'S LACTATE SOLUTION (HARTMANN)*

	gm/1	mEq/1			
Na lactate	3.1	Na	130	Cl	109
NaCi	6.0	K	4	Lactate	28*
K Cl	0.4	Ca	3		
Ca Cl_2	.2				

The majority of children will respond well to the above phase of management. There are some infants who have a disproportionate loss of water to sodium from hyperventilation, fever and diarrhea. This state has been termed hyperosmolarity.[51,52] Central nervous system signs and convulsions are seen in this group. The skin has a firm velvety resistant texture. Treatment is modified to give more glucose, water and solutions containing less sodium. When a child has an extreme degree of sodium depletion as evidenced by marked loss of interstitial fluid and circulatory collapse and, if the determinations of serum show a lowered sodium (or $CO_2 + Cl + 10$). Ringer's lactate should be administered without the addition of 5 per cent glucose. No specific treatment for the metabolic acidosis is undertaken and the full advantage is taken of the regulatory power of the functioning kidney. The extracellular fluid is expanded by Lactate Ringer's solution and 5 per cent glucose water which contains sodium ions in excess of chlorine. The water intake is adequate so that diuresis occurs and the acidosis is gradually corrected.[53]

Harrison has shown after six years of experience that, "when laboratory data cannot be obtained rapidly and the clinical appraisal does not suggest usual sodium depletion or disproportionate water deficit this program of therapy is one of choice."[46] The author has used it with minor variations during the past four years and found that he is

---

*Metabolized to HCO_3
†See Ringer-Lactate Formula
**Lytren Formula

in agreement with these findings, and that with this system the minimal use of the laboratory has been required.

Children above the age of two years seldom require the intensive treatment of diarrhea as outlined above.[54] They are better able to maintain their homeostasis, despite their frequent loose stools. Usually an adequate fluid intake plus treatment with antibiotics is all that is required.

Summary

The primary purpose of presenting a paper to an assembly such as this is to further postgraduate medical education. The author has endeavored to use good pedagogic procedure in relating the well-known causes of diarrhea to those recently reported as causing diarrhea in children. To this end, the field of acute diarrhea as a manifestation of disease and the causative agents of acute diarrhea have been reviewed. Treatment of the acute diarrheal state has been outlined.

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"MISSILE AND SATELLITE EMPHASIS MUST NOT HOLD BACK MEDICAL RESEARCH"

A warning that the current preoccupation with missiles and satellites must not be permitted to hold back vital life-saving research in the medical sciences was sounded by Dr. Robert W. Wilkins of Boston, President of the American Heart Association, speaking before a luncheon meeting of the Advertising Club of Baltimore, held in January as a forerunner of the 1958 Heart Fund campaign being conducted during the entire month of February.

In his talk, Dr. Wilkins urged that "wholehearted support, not just lip service, be given to the biological sciences and the needs for research and education in this area so that we may break through the barriers of ignorance in our struggle against disease. In our preoccupation with military security and the physical sciences, we must not allow the medical scientist to assume the status of a forgotten man."

Dr. Wilkins said that basic research, which he defined as the search for knowledge without commitment to immediate application or cures, "holds the key to further advances in all fields." To the extent that basic research is adequately understood and supported, he said, "we shall go forward to ever greater mastery of the world in which we live," a mastery that includes the conquest of disease.

"Let me make it perfectly clear," Dr. Wilkins told his audience of advertising men, "that I join you, and all other good Americans, in the fervent prayer that our scientists will achieve whatever is necessary in order to protect the security of our nation and our way of life. But at the same time," he added, "I should like to express the hope that we do not permit our perspective to become blurred," with regard to the necessity of supporting the biological sciences.

Somewhere in the United States at this very moment, Dr. Wilkin said, there may be an "underpaid and unsung medical scientist" who may in time make as valid a claim to enduring fame as the developers of satellites and missiles by finding the exact causes for high blood pressure and hardening of the arteries. These disorders are responsible for about 90 per cent of all heart disease.

"The achievements of this medical scientist," Dr. Wilkins said, "will be reflected in the saving of millions of human lives, rather than in the perfection of methods of mass destruction. Since the diseases of the heart and blood vessels are now responsible for more than 800,000 deaths yearly in this country alone, or about 54 per cent of all deaths, the unidentified scientist of whom I speak will have done as much, if not more, to advance and benefit humanity than any of the nuclear or missile scientists."

In general, however, Dr. Wilkins said there recently have been signs that the "so-called awakening" to the true meaning of basic research is more apparent than real. "There are evidences that the tributes we hear are indeed mere lip service, based on inadequate comprehension of what basic research really means," Dr. Wilkins noted, and he added, "If the process of investigating *all* aspects of our world, through the physical and biological sciences, is to be held back by an insistence on immediate results in the arms race, we may again find ourselves back in the same boat. We shall be seriously behind in a field on which the welfare and survival of our people depend. This can be prevented if we truly acknowledge the scope of basic research and give it the financial encouragement it needs."

The Current Status of Hereditary Pancreatitis

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IN 1952 Comfort and Steinberg[1] reported the unusual occurrence of pancreatitis affecting four members of a single family and possibly affecting two others. At that time such familial incidence was unique, although Collett and Kennedy[2] in 1948 had reported the occurrence of chronic relapsing pancreatitis in a child with possible involvement of a sibling, and two years later Poulsen[3] had published a similar observation. In each of these two latter reports the child affected with pancreatitis and the sibling suspected of having the disease, exhibited hyperlipemia. Earlier this year we[4] reported the pedigrees and additional observations based on study of two other families with hereditary pancreatitis. In one of these families seven persons in three generations are affected, and pancreatitis is rather strongly suspected in seven of their blood relatives; in addition, another eight persons in this family have experienced atypical abdominal pains of some severity. Jackson[5] recently has encountered a family in which two brothers definitely have pancreatitis and their mother and a child of one of the brothers may be affected also; in this family hyperparathyroidism also prevails. In the past two years we have encountered at the Mayo Clinic several other families in which two or more members definitely have had pancreatitis.

Clinical and Genetic Aspects

What, then, are the clinical features of hereditary pancreatitis, and how does this form of the disease differ from nonhereditary pancreatitis? It should be noted at the outset that experience with hereditary pancreatitis is still small, so that any generalizations are necessarily of a rather tentative nature. Our experience to date indicates that hereditary pancreatitis resembles in most ways the nonhereditary form of the disease.[1,4,6,7] Hereditary pancreatitis is characterized by the same severe, prolonged seizures of pain in the upper abdomen and back, and in the course of time the resultant pancreatic destruction may be followed by any or all of the same sequelae: diabetes mellitus, pancreatic calcifications, external pancreatic insufficiency with steatorrhea and azotorrhea, and formation of pseudocysts.

Differentiation from Nonhereditary Pancreatitis.—Nevertheless, there appear to be certain differences between the hereditary and nonhereditary forms of pancreatitis. The first, of course, is the familial incidence in the hereditary form; not only are multiple members of a kindred affected, but such involvement seems to be in accordance with Mendelian laws in these families. A second, striking difference between the two forms of the disease is the onset early in life in hereditary pancreatitis. Whereas in the nonhereditary form of the disease the attacks usually begin in the fourth decade of life,[6,8] in most cases of hereditary pancreatitis the attacks begin in childhood. In our experience this onset early in life has been a very helpful diagnostic feature, so that we have learned to suspect rather strongly the hereditary form of pancreatitis when the patient is a child or young adult, or when the attacks date back to childhood. Under these circumstances a painstaking inquiry into the familial history is likely to be rewarded with the discovery of other such cases.

A few other clinical differences between the hereditary and the nonhereditary forms of pancreatitis deserve mention. So far, hereditary pancreatitis has affected females approximately one and one-half times as frequently as males, whereas in most reported series of nonhereditary pancreatitis males have outnumbered females by two or three to one, and in one series by six to one.[6,8] This apparent slight predilection of hereditary pancreatitis for the female may, however, reflect

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only chance variation in an admittedly small sample. It is interesting that among the cases of hereditary pancreatitis there has been none so far with associated gallstones or disease of the biliary tract. Also of interest, as was observed in the family reported by Comfort and Steinberg,[1] is the fact that the pancreatic calcifications encountered among cases of hereditary pancreatitis have been almost exclusively calculi in the larger pancreatic ducts. Hyperlipemia has been observed only once, and alcoholism has been conspicuously absent from cases of hereditary pancreatitis, although a few of the patients stated that they had formerly used alcohol but had for some time abstained completely, since alcohol had precipitated attacks of abdominal pain.

Although hereditary pancreatitis has been recognized rather infrequently to date, it seems likely that cases will be encountered more frequently as acquaintance with the entity increases. It has been instructive to note the frequency with which bouts of pancreatitis have masqueraded as duodenal ulcer, gastritis, gallstone colic, and other similar entities in the hereditary cases.

Heritability.—As yet, the genetic observations are somewhat tentative also, and are based partly on the presumption that most of those persons in the affected families now only suspected of having had attacks of pancreatitis eventually will prove to have the disease. Such a presumption appears not unjustified, since the description of the attacks of abdominal pain in most of these persons is characteristic of pancreatitis. The available evidence indicates that the mechanism of inheritance is a nonsex-linked Mendelian dominant gene.[1,4] It is not yet settled whether the phenomenon of poor penetrance sometimes is involved; in such instances it is postulated that the person in question has inherited the abnormal trait, perhaps in heterozygous fashion, although without development of the clinical manifestations of the disease.

Etiologic and Pathogenic Considerations

The etiology and pathogenesis of chronic relapsing pancreatitis are unknown. In hereditary pancreatitis Nature seems to have provided us with an unusual opportunity and challenge, for the development of the disease in accordance with Mendelian laws in some families and the appearance early in life in the person affected make it appear virtually certain that they have inherited some predisposing abnormality. Although the evidence now available is rather meager, it seems that such an inherited abnormality is more likely metabolic or biochemical than anatomic in nature.

In an effort to obtain evidence which might bear on the nature of the presumed inherited defect predisposing to hereditary pancreatitis, we[9] assayed, microbiologically, the apparent free and total (that is, free and combined) forms of fourteen individual amino acids in fasting serum and twenty-four-hour urine specimens from two groups of persons: in the first group were seven persons with hereditary pancreatitis and four of their blood relatives apparently without the disease; in the second group were eleven persons with the nonhereditary form of pancreatitis.

Amino Acid Abnormalities.—Certain patterns of the serum and urinary amino acids observed in both groups with pancreatitis appeared abnormal when the data were compared with those obtained previously[9-11] in study of healthy persons. Further, the patterns of abnormality were somewhat different for the hereditary and the nonhereditary groups. The most distinctive and outstanding abnormality encountered in the patients with hereditary pancreatitis was excessive urinary excretion of lysine. Excessive excretion of leucine occurred in both groups. This amino-aciduria (lysinuria) was observed in multiple members and generations of several families with hereditary pancreatitis, and in one seemingly healthy blood relative in each of three such families (two of these relatives being children). It is of particular interest that the phenomenon of lysinuria was largely confined to the group with hereditary pancreatitis. The amino-aciduria did not seem to be related to the state of activity of the pancreatitis when such existed or to the presence or absence of diabetes mellitus.

Quantitatively, the urinary losses of amino acids were comparable to those which have been found by others in cases of diabetes mellitus, pneumonia, and hepatic disease.[12-15] The largest excretion of lysine was observed in one case of hereditary pancreatitis with general amino-aciduria; in this case the losses were approximately 25 per cent as large as those which have been described in cases of cystinuria, hepatolenticular degeneration, and Fanconi's syndrome.[14-31]

Lysinuria has been described as part of a more or less general amino-aciduria in a number of conditions, including Wilson's disease and Fanconi's syndrome, and is also an integral part of the more limited amino-acidurias of cystinuria and pernicious anemia in relapse.[18,21-23,29-38] However, in these other conditions, except for cystinuria, in which the urinary excretion of lysine commonly exceeds that of cystine,[29] the excessive urinary excretion of lysine is not the major amino acid defect observed, as it appears to be in hereditary pancreatitis.

The pathogenesis of the observed abnormalities of amino acids in serum and urine of persons with pancreatitis is not clear. The data do not justify exclusion from consideration of either the overflow mechanism or impairment of renal tubular reabsorption as possible factors in the pathogenesis of the observed amino-aciduria; it may be that both factors play some role. It is possible that the seemingly distinctive amino-aciduria in the cases of hereditary pancreatitis reflects an inborn error of protein metabolism, as occurs in phenylketonuria[38-42] and perhaps in Wilson's disease.[25,26,43,44] It is to be hoped that renal clearance studies of lysine and leucine in these cases may provide information which will serve to answer some of the still unanswered questions.

Porphyrin Abnormalities.—Brief mention may be made of certain other observations which also, unfortunately, at present serve only to raise more perplexing questions. In one of the families with hereditary pancreatitis the authors[4] encountered weakly positive results for porphobilinogen on qualitative testing of urine of one man with definite hereditary pancreatitis and sequelae and that of two of his nephews, one of whom had experienced attacks of abdominal pain characteristic of pancreatitis. Both nephews (brothers) exhibited the unusual amino-aciduria characterized by excessive excretion of lysine and, in fact, had almost identical patterns of amino acids in serum and urine. Again, in the case of a young man of another family with hereditary pancreatitis a weakly positive qualitative result for urinary porphobilinogen was encountered. This person had experienced classic attacks of pancreatitis over a period of ten years and had passed red urine on one occasion several years prior to the study. Analysis of his feces disclosed slightly elevated values for fecal coproporphyrin and protoporphyrin. One hesitates to attach much importance to the borderline porphyrin abnormalities observed; and even if these should prove significant, any possible relationship between such porphyrin abnormalities and the pancreatitis, or between these abnormalities and the amino acid abnormalities observed, remains conjectural at present.

It is of interest in this regard, however, to note that Berger and Goldberg[45] recently have reported the association of amino-aciduria and excessive excretion of coproporphyrin III and some intermediate porphyrins in the stool and urine of a boy, both his parents, and a paternal aunt, and have termed this condition hereditary coproporphyria. The amino-aciduria in these cases was characterized by what was described as a "super-glycine pattern."

The association of amino-aciduria and porphyrin abnormalities was also described last year by Baron and associates,[33,46] in what they have designated as "H, or Hartnup, disease." In this interesting syndrome, which also seems to be inherited, four siblings of eight from a marriage of first cousins exhibited a tendency to develop a pellagra-like rash on exposure to sunlight, sometimes associated with severe cerebellar ataxia and mental retardation. The constant amino-aciduria in Baron's cases appeared to be of renal origin and of a unique pattern, which included moderately increased excretion of lysine as determined chromatographically. The feces of these persons were found to contain moderately increased quantities of protoporphyrin.

Whether the association of abnormal excretion of porphyrins and amino acids observed to date is more than coincidental remains to be determined. It may be appropriate here to mention also the two cases reported by Saint and associates[47] in which porphyria and pancreatitis coexisted; these authors suggested that the pancreatitis had resulted from focal ischemia as a consequence of porphyric involvement of the smooth muscle in the arterioles of the pancreas.

Association with Hyperparathyroidism. — The interesting family described by Jackson,[5] in which parathyroid adenomas and hyperparathyroidism had developed in multiple members, two of whom were said to have experienced recurrent bouts of pancreatitis, brings to mind the possibility of some inherited defect having to do with calcium metabolism in those who exhibit the hereditary form of pancreatitis. In recent years there have been sporadic reports of pancreatitis and hyperparathyroidism coexistent in the same person, and the number of such reports seems to be growing.[48-52] In such cases it usually has been postulated that the hyperparathyroidism has resulted in the formation of stones in the pancreatic ducts, with the development of pancreatitis secondary to this ductal obstruction. In our own cases of hereditary pancreatitis the evidence to date seems to point away from the possibility that the pancreatitis was secondary to hyperparathyroidism. The values for serum calcium obtained from some of these persons between attacks have not been elevated; and in a number of instances the pancreatitis has been unassociated with pancreatic calcifications, or has preceded the development of such calcifications by some years.

Treatment

In the present state of our knowledge the management of cases of hereditary pancreatitis is the same as for cases of nonhereditary pancreatitis. Prophylactically, the physician has little to offer such patients except avoidance of alcohol and spices. Nonsurgical management otherwise consists of supportive measures and replacement therapy. The surgical approach, although still leaving much to be desired, has more to offer such patients but must be individualized. In general, it has seemed best to avoid operating on the children and young adults who have hereditary pancreatitis until the trend, tempo, and severity of the disease make some form of surgical intervention seem clearly advisable.

Summary

In some families, pancreatitis seems to be inherited via a Mendelian dominant gene, nonsex-linked. Hereditary pancreatitis almost always begins in childhood or early adult life. It is characterized by the same severe, prolonged seizures of abdominal pain as mark the nonhereditary form. To date, hereditary pancreatitis has been found to occur somewhat more frequently in females and has been unassociated with gallstones, hyperlipemia (one case excepted) or alcoholism.

It appears likely that persons with hereditary pancreatitis have inherited some predisposing abnormality, perhaps of a metabolic nature. Microbiologic assays have revealed certain abnormalities of amino acids in the serum and urine of persons with hereditary pancreatitis and in a few instances in their blood relatives not affected with pancreatitis thus far; these amino acid abnormalities are somewhat different from those observed in persons with nonhereditary pancreatitis. The amino-aciduria of hereditary pancreatitis is rather distinctive in that there is excessive excretion of lysine. The observed abnormalities of amino acids may signify some defect in intermediary protein metabolism. Whether the amino-aciduria may stem, at least partly, from impairment of renal tubular reabsorption is not yet known. Borderline porphyrin abnormalities have been encountered in a few persons with hereditary pancreatitis and in one blood relative without pancreatitis; the significance of this observation, if any, is not now apparent. To date none of the cases of hereditary pancreatitis observed by the authors has been found to have coexistent hyperparathyroidism. The treatment of hereditary pancreatitis at present is the same as that for the nonhereditary form of the disease.

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Pathogenesis of Collagen Diseases

A General Review with Emphasis on the Face and Head

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THE UPPER part of the respiratory tract extending from the lips and nasal vestibule to the larynx has not ordinarily been considered as a site of collagen disease. Semenov,[1,2] however, stated that certain disorders of the tunica propria of the nasal mucous membranes, particularly the type usually termed "hyperplastic sinusitis," should be included in the category of collagen diseases. He quoted Maximow and Bloom as saying that there are four main types of connective tissue, all characterized by abundant intercellular substance: (1) blood and lymph, (2) connective tissue proper, (3) cartilage and (4) bone.

In the connective tissue proper, the intercellular substance always contains fibers and varies from a soft jelly to a tough fibrous mass. Klemperer and associates[3] used the term "collagen system" to refer to the fiber-forming connective tissues throughout the body. Such tissue occurs in the nose and throat, particularly in the nasopharynx, the reticulum of tonsils and adenoids, and laryngeal polyps. One of the most obvious examples of collagen disease according to Semenov is nasal polyposis. He stated that polyps, whether bacterial or allergic in origin, have a poor blood supply and a tremendous amount of edema, which begins with abnormal capillary permeability. Perennial allergy or chronic infection or both produce profound effects in the collagen substance and perpetuate the edema. Semenov also said that polypoid sinusitis is characterized by a tremendous amount of chronic edema or a lesser degree of edema with thickening and degeneration of collagen tissue, hyalinization of the basement membrane and infiltration by eosinophils, lymphocytes and plasma cells.

Although allergy is a common cause of polypoid sinusitis, it is not the only cause. Infection may lead to similar changes. Histologically, edema and increase in collagen substance, hyperplasia of connective tissue, and infiltration of leukocytes, eosinophils, plasma cells and lymphocytes may be seen in various proportions in the mucous membrane in polypoid sinusitis. The actual hyperplasia becomes evident when the edema subsides. Even though the pathologic changes are divided into exudative, proliferative and degenerative stages, the main feature is the amount of permanent thickening.

Semenov[1,2] has presented convincing evidence that the collagen tissues of the nose and throat may be involved in disease processes and that these disease processes are frequently allergic. This is not exactly the same as classifying the disease processes as collagen diseases or as demonstrating that the collagen dieases are, as he implies, frequently allergic in origin.

To clarify these points it appears necessary (1) to consider what Klemperer had in mind when he first presented the term "collagen diseases," (2) to follow the development of the concept of collagen disease and (3) to determine what is meant by the term "allergy." It is helpful also to review briefly the available information on the ground substance of connective tissue, with its fibers and fibrils.

Definition of Collagen Disease

Klinge,[4,5] in 1929 and 1933, introduced the concept that a group of systemic diseases affects connective tissue. He focused attention on the conspicuous changes of the intercellular components of connective tissue, the fibrinoid alteration of collagenous tissue and the myxomatous swelling of the ground substance. Observation of similar changes in connective tissue in animals made hypersensitive to foreign proteins led Rössle[6] and Klinge[5] to conclude that the tissue damage in rheumatic fever and rheumatoid arthritis was

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caused by hypersensitivity. Klinge stated that the same pathogenic mechanism was present in periarteritis nodosa, dermatomyositis, malignant nephrosclerosis, thromboangiitis obliterans, certain nephritides and cardiovascular sepsis.

Klemperer and co-workers[3] suggested the collective term "collagen diseases" to indicate the site of the characteristic lesion in these diseases, namely fibrinoid necrosis of the collagen fibers. Klemperer[7] later stated that when he and his associates originally proposed the term "collagen diseases" they were aware that the structural alterations which had been disclosed by conventional techniques of microscopic anatomy required further analysis. He considered that the term was not applicable to diagnosis and certainly did not apply to all the morbid processes of these diseases; all he wanted to express originally was that anatomic investigations in certain diseases disclosed conspicuous systematic changes in the intermediary substances of the connective tissue. Klemperer[8] also stated that a biologico-anatomic synthesis so sweeping as to include the collagen diseases among the allergic maladies was premature. He referred to the observations of Byron and Dodson, who produced fibrinoid necrosis in the arteries of normal animals by brief increases in intra-arterial tension. A number of other workers also have produced collagen diseases in animals by methods that probably excluded the factor of hypersensitivity.

Klemperer[7] regarded the connective tissue as the common seat of these heterogeneous maladies. He did not deny the probable role of hypersensitivity but was interested in a search for all factors that might be responsible for the conspicuous structural alterations; he also was interested in the mechanism of their action. It appeared obvious that such an inquiry had to take its origin from existing information concerning the structure and biology of normal connective tissue. He pointed out two problems that have yet to receive an unequivocal solution, namely the mode of fiber formation, and the nature and site of fabrication of the homogeneous ground substance.

Ground Substance and Fibers of Connective Tissue

Connective tissue is made up of an amorphous ground substance, mucinous in nature, containing collagen fibers. As Duran-Reynals[9] stated, the ground substance of the mesenchyme is an entity as old as histology itself, although it now may appear as a new one. The mesenchyme is likely to be considered an inferior structure that serves, depending on the circumstances, as a support to the passageway or as a lubricant. In the present concept, the ground substance is considered a part of other structures but as having also its own physiognomy and as functioning as a coherent unit.

The ground substance is a plastic material existing apparently in the form of a gel. In young undifferentiated connective tissue, ground substance is abundant and continuous; in adult subcutaneous tissue, it is less plentiful, being concentrated about the collagen fibers. The gel is greatly hydrated and therefore involved in water binding. Bensley[10] stated that the intercellular ground substance of loose connective tissue is a continuous, fairly granular, transparent material in which fibers are imbedded. It is elastic and tends to retract when cut. It is viscous and possibly acid. It is digested by trypsin but not by pepsin; the reverse is true of collagen. It combines with copper salts, suggesting that it may contain a derivative of chondroitin sulfuric acid.

Vaubel[11] found a synovioblast that apparently secreted mucin and was the source of the ground substance. He found also that secretion of mucin diminishes when synovioblasts change into fibroblasts. The mucinous nature of the homogeneous ground substance has been known for decades.

Kling[12] considered that the production of mucin was the result of cellular activity and that in inflammation the number of cytoplasmic granules in the synovioblasts of Vaubel and the secretion of mucin increase greatly.

Volterra[13] demonstrated that the adventitia is an indispensable element of all capillaries. Impregnation with silver shows that the adventitia consists of a delicate reticulum spread on and merged with an amorphous fundamental substance; the whole structure forms a complete membrane composed of what Volterra called "lamellated reticular tissue," which is continuous with the fibers of the surrounding connective tissue. Chambers and Zweifach[14] noted that the cement present between the endothelial cells of the capillaries is a sticky argyrophilic substance, continuously secreted by the cells themselves and conditioned by calcium salts and the pH. They found that the cement behaves as a salt of calcium, that it is digested by trypsin and that it is not laid down in the absence of vitamin C.

Duran-Reynals[15] stated that the permeability of the connective tissue can be increased by certain factors and decreased by others. He found that the most important effect in increasing this permeability is that of the spreading factors, some of which are identified with mucolytic enzymes.

McMaster and Parsons[16] injected vital dyes into the connective tissues and noted that in the absence of edema the dye escapes from the lymph vessels as bristly, wavy lines of color formed by the dye moving between or along the fibrils of the connective tissue. The fluid moves through the tissues in thin films "captured" by capillary forces as if caught between two pieces of glass. As Duran-Reynals[15] pointed out, despite the viscosity of the ground substance, metabolites must move easily through it, impelled by capillary forces.

The investigations of Meyer[17] have greatly advanced knowledge of the mucopolysaccharides that enter into the constitution of the ground substance. They are mainly hyaluronic acid and chondroitin sulfuric acid. Meyer and Palmer[18] found a polysaccharide in the vitreous humor of the eyes of cattle consisting of hexosamine (d-glucosamine), hexuronic acid (glucuronic) and an acetyl. They named this product "hyaluronic acid." Meyer[17] suggested as a tentative feature of the development of cement substance in mesodermal tissues that the young fibroblast secretes hyaluronic acid, a precursor of collagen, and a chondroitin sulfate. By local acidification in the immediate neighborhood of the cells, the first fibers are produced by the polysaccharides from the native soluble collagen, which denatures into the soluble fiber, on the surface of which lies a sheet of polysaccharides. With aging, the polysaccharide layer becomes thinner and the hyaluronic acid is replaced more and more by chondroitin sulfate. Only in metabolically active tissues like that of skin does production of hyaluronic acid continue in appreciable quantities.

The fibers of connective tissue are divided into collagenous, elastic and reticular types. Studies with the electron microscope have shown that connective tissue contains many more fibers than was previously suspected. Even the smallest collagen fibers visible with ordinary microscopes are composed of bundles of minute fibrils, each of which exhibits periodic cross striations when viewed with the electron microscope.

Klemperer[8] pointed out that, while many experiments suggest that both the ground substance and the fibers of connective tissue are the result of cellular activity, the possibility that the ground substance is a transudate from the capillaries and that fibrin and collagen fibers can be formed from plasma clot by mere mechanical factors has not been completely ruled out.

Hass and McDonald[19] stated that an interaction between fibroblasts and ground substance resulted in formation of collagen. Robertson and Schwartz[20] confirmed the results of numerous histologic studies demonstrating the necessity of ascorbic acid for formation of collagen. They also found that the rapid formation of tissue containing large amounts of collagen increased the nutritional requirements of guinea pigs for ascorbic acid. The ascorbic acid in the process of fiber formation may be a component of chondroitin sulfate and may replace in the chain some of the molecules of glucuronic acid.

Ehrich[21] stated that the production and maintenance of connective tissue depend not only on the activity of fibroblasts and of the import of building stones from the blood, but also on regulating principles, such as enzymes, vitamins and hormones. The enzymes that degrade hyaluronic acid and chondroitin sulfuric acid are the hyaluronidases. They appear to be mixtures of several enzymes, some depolymerizing the long-chain molecules, others hydrolyzing aldobionic acid. The action of hyaluronidases *in vivo* is conditioned by the presence of antihyaluronidases. Ascorbic acid is outstanding among the vitamins that control connective tissue. Ehrich noted that thyrotropic hormone can stimulate the production of both ground substance and collagen fibers. However, he found that the action of adrenocorticoids had special effect.

Pathologic Physiology of Collagen Diseases

In discussing the pathology of connective tissue, Ehrich[21] stated that the alterations are degenerative or proliferative. The degenerative changes are mucoid degeneration, fibrinoid degeneration and necrosis, amyloidosis and para-amyloidosis. The proliferative change is the secondary proliferation of connective-tissue cells, which is a common sequel of degeneration and necrosis of both connective tissue and granulation tissue.

Primary proliferation of connective-tissue cells occurs in myxedema and generalized scleroderma and probably in other collagen diseases. Ehrich found severe primary proliferation to be a characteristic feature of subacute allergic reactions (granulomas). Allergic proliferation is usually located in the intima or adventitia of blood vessels.

Ehrich also reviewed the pathologic physiology and morphology of each collagen disease, including rheumatic fever, rheumatoid arthritis, lupus erythematosus disseminatus, generalized scleroderma, dermatomyositis, serum sickness and periarteritis nodosa. He considered that rheumatic fever probably is an allergic response to either group A hemolytic streptococci or their products. He stated that the cause was unknown in rheumatoid arthritis, lupus erythematosus, generalized scleroderma and dermatomyositis. He remarked that it is well established that serum sickness is an allergic reaction due to intravenous injection of foreign serum or other foreign proteins and that the arterial changes in periarteritis nodosa resemble those in serum sickness so closely that the two appear to be the same; thus, he concluded that periarteritis nodosa is an allergic disease due to an Arthus reaction.

Long[22] stated that the collagen diseases are systemic, not local, conditions. Klinge[4] considered fibrinoid necrosis to be presumptive evidence of a hypersensitivity reaction. He thought that systemic diseases exhibiting fibrinoid necrosis have not been shown to occur in the absence of naturally occurring or artificially induced allergic reactions. The observation that fibrinoid necrosis may develop locally as a result of mechanisms other than allergy did not in Long's opinion disprove Klinge's hypothesis. Long stated that allergic reactions are of two types, namely immediate (anaphylactic), which is either systemic or local, and delayed, which is the result of bacterial allergy (tuberculin type); in his opinion, the delayed type of hypersensitivity reaction is the one present in collagen diseases. He stated that it is a normal sequence of any bacterial infection, so that it is not necessary to postulate an abnormal hypersensitivity reaction. It is a natural phenomenon in which sensitivity is induced by persistent low-grade infection. The response is delayed and is due to antibody fixed on the cells rather than to circulating antibody, as is the case in the immediate type of reaction.

Rich,[23] in discussing hypersensitivity in disease, stated that there is high promise in an attack on the virtually untouched fundamental problems relating to the intimate chemical mechanisms through which hypersensitive reactions exert their injurious effects on the tissues.

In a review of the possible allergic mechanisms involved in collagen diseases, Miale[24] stated that necrosing arteritis in man has been preceded or accompanied by bronchial asthma, a variety of allergic states, acute and chronic bacterial infections, protozoan infestation, administration of drugs and serum sickness. Since in only one or two of these situations is it possible to show qualitative and quantitative similarities to the Arthus experiment, it follows that vascular necrosis does not always represent an Arthus reaction but is more often an expression of other types of allergic reactions.

Rössle,[6] in observing by biomicroscopy the changes of "allergic inflammation" in the mesentery of the frog, noticed that the first reaction was in the blood flow. He found degenerative changes in the walls of small blood vessels, with deposits of fibrin in zones of edema. Following these changes, the fibrin was replaced by proliferative fibrocapillary tissue. Harkavy,[25] in pointing out the importance of the vascular reactions in the symptomatology of allergy, stated that when dealing with bacterial allergy the characteristic symptoms of the disease to which the patient is subject may be reactivated by contact with homologous bacteria responsible for the initial sensitizing effect, and also possibly by viruses and by heterologous bacteria and their toxins, as well as by antigen-antibody complexes of nonbacterial origin.

Several years ago, I[26] suggested that, because the allergic reaction made use of a stereotyped (invariable) vascular mechanism in producing its results and because an antigen-antibody component has not been demonstrated in the lesions of physical allergy, the immunologic mechanism might be supposed to produce so-called allergic responses without an antigen-antibody reaction being present. I thought that this might explain the infrequency of favorable clinical results from attempts at hyposensitization.

From a review of the literature, it is apparent that knowledge of the mechanisms of conditions considered to be allergic is still in its infancy. The difficulty that we physicians face when we treat patients with allergy is that we are trying to apply to clinical medicine a concept that is still in the stage of investigation. The concept of physical allergy presented by Weed never has been successfully controverted. Interesting hypotheses have been presented recently in which the altered reactivity in "allergy" is considered as not due to an antigen-antibody reaction but rather to an enzymatic disorder.

Jaros,[27] in presenting a hypothesis on the physiochemical pathogenesis of the collagen diseases, stated that availability of purified adrenocorticoids has unfolded a new era in medicine. In evaluating the pharmacologic effectiveness of such compounds, he considered that it was immediately apparent that epinephrine, acetylcholine and histamine are involved. The relationship of these three substances to the enzymes and to adrenocorticoids formed the basis for a new hypothesis. He emphasized that the abnormal mechanism involved apparently was present only in persons who had inherited a predisposition to collagen diseases. It was his belief that the tissues in such persons have an abnormal responsiveness, which is exhibited by exaggerated reactions toward acetylcholine and histamine formed in the sensitized end-organs. He suggested that the pathologic reaction and the attempt at adaptation occur in two phases. The catabolic phase concerns the deleterious effects produced in the tissues and is due to the incompletely antagonized acetylcholine and histamine, whereas the anabolic phase involves the adrenocorticoids, which enhance the formation of enzymes that inhibit or neutralize the excess of acetylcholine and histamine present. He suggested that the best management of the collagen diseases should include the use of cholinesterase or other efficient antiacetylcholine and antihistaminic drugs.

Godlowski[28] suggested that the manifestations of anaphylaxis (allergy) are not directly caused by the antigen-antibody interaction. He suggested that in the enzymatic concept of anaphylaxis the anaphylactic phenomena should be regarded rather as resulting from the failure of the antigen-antibody reaction.

Collagen Diseases of the Face and Head

From this review of the literature, it appears that hyperplastic sinusitis cannot be classified as one of the collagen diseases described by Klinge and by Klemperer. Although the proliferative and degenerative changes in the tunica propria seen in the collagen diseases are present in hyperplastic sinusitis, it hardly appears warranted to consider it as a generalized disorder, even though, according to Semenov, it often may be due to the bacterial type of allergy.

However, two disorders occur in the region of the head, one in the upper part of the respiratory tract and the other involving the eye and inner ear, that meet the demands of strict classification as collagen diseases. These are lethal granulomas of the midline facial tissues, and nonsyphilitic interstitial keratitis and deafness (Cogan's disease). In these conditions, the primary lesion is associated with generalized periarteritis nodosa.

Krompecher[29] reviewed the literature on destructive midline lesions of the face. His description of the pathologic changes is one that might correspond to what is now known as syncytial cell sarcoma. Kraus,[30] in 1929, described three cases of a "special neoformation" of the nasal cavities and suggested that destructive midline facial lesions might be granulomatous rather than malignant. MacNaughton-Jones in 1897,[31] apparently was the first to describe a granulomatous lesion of the midline facial tissues. In 1931, Wood[32] presented the first case of malignant granuloma reported in the North American literature. Wegener,[33] in 1939, described the association of midline granuloma with periarteritis nodosa, an association that since has been confirmed on numerous occasions.

Terracol and Camps,[34] in a scholarly and critical review of the literature, analyzed reports of seventy-four cases. They assumed that twenty-four of these lesions were due to an infectious process and twenty-one were malignant tumors, of which fourteen were reticulum cell sarcomas and seven were lymphosarcomas; six lesions were "malignant reticulosis" and sixteen were caused by an allergic affection. They considered that malignant granuloma ought not to be considered as an idiopathic disorder but as a syndrome of multiple causes.

Di Pietro[35] reported a case of nasal granuloma from which Rhinosporidium seeberi was isolated. De Faria and co-workers[36] reported three lesions resembling malignant granuloma that showed a histologic picture of reticulosarcoma. They suggested that all cases of "malignant granuloma" were of this type. However, none of the lesions for which a definite cause could be proved were associated with generalized periarteritis nodosa.

Midline ulcerations not associated with the generalized lesions of periarteritis nodosa would appear usually to be of a different cause than the true malignant granuloma with generalized periarteritis nodosa, although the local clinical appearance may be the same.

Because of the association of certain of these midline facial lesions with periarteritis nodosa, it was suggested by Williams and Hochfilzer[37] that administration of anti-inflammatory corticoids might reverse the process. Moore and associates[38] reported a case in which use of cortisone completely cleared up the granulomatous lesions in the nose, palate and pharynx. Although the granulomatous lesions did not recur, the patient died a year later from cardiovascular renal disease considered to be the result of healing of the lesions of the associated periarteritis nodosa.

Hagens and co-workers[39] reported a case in which malignant granuloma of the nose and face was treated with corticotropin (ACTH) and has remained healed for four years.

Vaheri[40] reported a malignant granuloma in a sixteen-year-old girl. Numerous eosinophils were found on biopsy of the lesion, and the patient had a blood eosinophilia of 10 per cent. The lesions involved the left orbit, left posterior pillar and left tonsil. Rapid improvement with use of cortisone was noted.

Summary

Review of the literature indicates that the reactions of connective tissue, especially the ground substance of the mesenchyme and the collagen fibers, are of great importance in certain diseases of a generalized type in which proliferative and degenerative changes occur in these tissues, especially in the walls of the blood vessels.

These reactions are stereotyped and invariable and suggest a common mechanism, which may be anaphylactic or allergic, particularly of the bacterial type. However, these reactions can be found when an antigen-antibody reaction cannot be demonstrated. Therefore, if these conditions are to be thought of as allergic, allergy must be considered in its broadest sense as an "unusual reaction" and not strictly as an antigen-antibody reaction.

The only conditions in the region of the head that strictly fit the definition of collagen disease are lethal granuloma of the midline facial tissues, and the syndrome of nonsyphilitic interstitial keratitis and deafness. However, polypoid hyperplasia with collagenous degeneration of the tunica propria of the nasal mucosa, although not associated with a generalized reaction, is a similar type of reaction and is known to respond favorably to cortisone and corticotropin. It is usually an allergic reaction of connective tissue, although it is not a "collagen disease."

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DETECTION OF POLYPS, POSSIBLE FORERUNNERS TO CANCER

By the early detection of small growths, called "polyps," in the large intestine, radiologists can help reduce deaths from intestinal cancer. The polyps are apparently often the forerunners of cancer. Once detected they can be removed surgically.

This was the comment of Dr. J. Maurice Robinson, San Francisco radiologist, in the April, 1957 issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

Dr. Robinson described x-ray procedures for these sometime obscure growths. He believes the procedure helps detect them more effectively.

"It has seemed evident that the problem of reducing the mortality of cancer of the large bowel and rectum, now responsible for 17 per cent of all deaths from cancer, is closely linked to the detection and eradication of the polyp, which is apparently the precursor of the malignant," Dr. Robinson explained.

After describing in detail the technical aspects of x-ray diagnostic procedures, Dr. Robinson added this warning: "The author has been forced" to the unhappy conclusion that there are unavoidable pitfalls inherent in any of these methods. The procedure which works well on one day will not work well on another; and on some days, none of them seems to be any good."

Other conclusions:

—One important element in assuring moderate success in finding these tricky growths is the earnest conviction on the part of the diagnostic radiologist that it is *important* to find them.

—Concentration of attention should be on the "happy hunting ground" which is the remote third of the large bowel. This is where 70 per cent of the polyps are located, in patients over fifty years old.

Even before the polyps have produced such physical symptoms as bleeding, radiologists have been able to locate them, previous reports indicate. "They appear to be punched out, as though with a cookie-cutter, and they seem to stand out much more sharply than one would expect. . . . Not infrequently the polyp defect shows as a very dense black center, the remainder of the defect being somewhat less dense."

Cooperation of Community Blood Banks in Supplying Blood for a Sensitized Hemophiliac Patient

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THE ROLE OF community blood banks in supplying blood for the everyday needs of patients in hospitals they serve, is well understood. Little or no difficulty is experienced ordinarily in providing an adequate supply of fresh blood or blood derivatives for the average patient. A real problem in blood procurement may present itself, however, if a patient having an unusual blood-group formula requires blood in large amounts. At such a time there is a great advantage in being able to call upon other blood banks for assistance in the specific problem encountered rather than to rely on an appeal to the public by radio, television or the press, which at best yields only an assortment of unselected bloods. The present report describes the satisfactory management of a problem that occurred at the Minneapolis War Memorial Blood Bank in supplying blood and fresh frozen plasma for a sensitized hemophiliac patient with a bleeding ulcer. The case is cited as an example of what can be accomplished when several community blood banks work together.

Case History

A thirty-nine-year-old man was admitted to a Minneapolis hospital on December 8, 1955, because of bleeding from a gastric ulcer. During the prior eighteen years, the patient had been hospitalized many times for various illnesses and injuries, which included trauma to the knees and other joints, dental surgery, appendectomy and five episodes of bleeding from the gastric ulcer. He had received between thirty-five and forty transfusions, several of which had caused reactions. During these many hospitalizations, a diagnosis of hemophilia had been made.

During the current admission, studies of the patient's plasma showed a prolonged plasma coagulation time, which was greatly shortened by the addition of fresh normal plasma and of plasma treated with barium sulfate, but which was not influenced by the addition of fresh serum.[1] Attempts at crossmatching at the hospital indicated the presence of abnormal blood-group antibodies. The patient's blood was sent to the Minneapolis War Memorial Blood Bank for cross-matching and studies.

Serological Findings.—The blood-group formula of the patient for the factors tested was group 0, Rh-positive (probable genotype R^1R^1 [CDe/CDe]), P-, K-, Fy(a-). It was apparent that he lacked rh″(E), hr′(c), P, K and Fy(a+), to any one or more of which factors he could have been sensitized. The significant reactions of patient and donor test cells to antisera and of the patient's serum to these cells are shown in Figure 1.

The results in this table showed that the patient had multiple antibodies: anti-hr′(anti-c), anti-rh″(anti-E) and anti-Duffy(anti-Fy^a). Antibody titers were determined on the patient's serum. It contained anti-hr′(anti-c) in a titer of 2 in saline, 32 in albumin and 64 by the indirect Coombs' test. The anti-rh″(anti-E) was demonstrable in albumin in a titer of 1 and by the indirect Coombs' test in a titer of 4. The anti-Duffy (anti-Fy^a) titer was 2 in saline, 1 in albumin and 4 by the indirect Coombs' test. A probable dosage effect was observed in the early specimens. Duffy cells were agglutinated in saline, whereas others were demonstrable only by the indirect Coombs' test; the latter were probably heterozygous. Evaluation of the requirement of antihemophilic globulin indicated that clotting times were normal at intervals of one, five and eleven hours after administration of 100 cc. of fresh frozen plasma.

Medical Management

The medical management of this present episode of gastric bleeding was successfully accomplished without complications and without transfusion reactions. At this point, a decision relative to definitive surgical treatment had to be made. The situation presented a unique problem in evaluating relative risks. This patient, a hemophiliac now thirty-nine years old with a history of five previous episodes of gastric bleeding, faced a progressively greater risk of future medical or surgical failure as his hemophilia became complicated by arteriosclerosis. If his future management was dependent on blood transfusions and fresh plasma, the possibility existed that he would not only acquire new sensitivities to other blood antigens but also acquire antibodies against the antihemophilic globu-

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From the Minneapolis War Memorial Blood Bank and Mt. Sinai Hospital, Minneapolis, Minnesota.

lin itself. Finally, because of the already existing multiple sensitizations, there was the great problem of supplying the specific blood type in amounts adequate to control a severe acute episode of bleeding wherever, geographically, the next emergency occurred. Since about 17 per cent of Western Europeans are homozygous rh′ (C) and homozygous hr″ (e), and about 30 per cent are Duffy (Fy^a) negative, one could expect to find about 5 per cent of group O bloods to be compatible with the patient. In Minnesota, about 40 per cent of bloods belong to group O; therefore, about 2 per cent of blood donors selected at random would have the righ blood formula to be compatible with that of the patient.

SIGNIFICANT REACTIONS* OF CELLS AND PATIENT'S SERUM

Anti-sera											Patient's serum				
											Crossmatches				
Cells	Blood Group	Rh_o D	rh' C	rh" E	hr' c	hr" e	P	K	Fy^a	Papain Screening	Saline 37°	Saline 20°	Albumin 37°	Albumin 4°	I.C.T.
Patient L.S.	O	+	+	−	−	+	−	−	−		−	−	−	+ −	−
R.H.	O	+	−	−	+	+	+	−	+	4+	+	+	4+		4+
R.S.	O	−	+	−	+	+	+	−	−	4+	+	+	4+		4+
R.K.	O	−	−	+	+	+	+	+	+	4+	+	+	4+		4+
F.H.	O	−	−	−	+	+	−	−	−	4+	+	+	4+		4+
8196	O	+	+	−	+	+	−	−	−		+	+	4+		4+
26470	O	+	+	−	−	+	+	−	+		+	2+	+		3+
8440	O	+	+	−	−	+	+	+	+		−	−	−		3+
8399	O	+	+	−	−	+	+	+	−		−	−	−		−

*Reactions of cells to antisera M, N, S, s, Le^a, Le^b, k, Fy^b, Lu^a, and Jk^a are known for some of the bloods on the table, but these are not significant and therefore not shown.

Fig. 1. Reactions in a hemophiliac patient undergoing gastrectomy. I.C.T. indicates indirect Coombs' test.

After weighing these risks against the well-known dangers of doing a major surgical operation upon a hemophiliac patient, it was the opinion that elective surgical intervention in the interval between bleeding episodes was the lesser risk. Accordingly, gastric resection was done on December 26. Six units of compatible whole blood were found in the bank's entire inventory and these, together with large amounts of fresh frozen plasma, were on hand at the time of operation. Hemostasis at operation was meticulous. The smaller bleeding points were controlled by coagulation and the larger ones by ligation. Nevertheless, the incision, though apparently dry when closed, soon began to bleed continuously. On the second postoperative day, a very small cutaneous bleeder was ligated after spreading the incision for 2.5 centimeters. Gastrointestinal bleeding was evident from the ninth to the fourteenth postoperative day and again on the twenty-third day. On the twenty-sixth day after operation, disruption of the middle third of the wound occurred. Little evidence of healing was present, the wound having the appearance of one separating on the seventh or eighth day. No evidence of infection or wound hematoma was noted. The wound surfaces appeared healthy, but little fibroplasia was evident. The disruption was sutured, but the healing continued to be slow. The retention-suture holes bled frequently, and the wound was not healed firmly until after a period of five months.

Administration of Blood and Fresh Frozen Plasma.—Fresh frozen plasma and whole blood were given for eight weeks and the patient was hospitalized for nine weeks. During this time, the patient received plasma in increasing amounts

TABLE I. BLOOD AND PLASMA ADMINISTERED TO A HEMOPHILIAC PATIENT

Date	Postop. Day	Plasma cc.	Blood cc.	Comment
12-24		100		
12-25		200		
12-26		150	1900	Gastrectomy
12-27	1	585	1050	
12-28	2	400	1800	Wound bleeder caught
12-29	3	500	500	
12-30	4	500	500	
12-31	5	500		
1- 1	6	600		
1- 2	7	500	2000	
1- 3	8	900	500	
1- 4	9	1200	1450	G. I. Bleeding
1- 5	10	1200	2900	
1- 6	11	1200	1500	
1- 7	12	1100	1500	
1- 8	13	1200		
1- 9	14	1200	500	
1-10	15	1200		
1-11	16	1200		
1-12	17	1200		
1-13	18	1200		
1-14	19	1150		
1-15	20	650		
1-16	21			
1-17	22			
1-18	23	200		G. I. Bleeding
1-19	24	400	1000	
1-20	25	800	1500	
1-21	26	1200	2500	Wound separation
1-22	27	1500	3000	
1-23	28	1800		
1-24	29	1425		
1-25	30	1250		
1-26	31	1500		
1-27	32	1000		
1-28	33	1000		
1-29	34	900		
1-30	35	700		
1-31	36	1500		
2- 1	37	1200		
2- 2	38	1250		
2- 3	39	1150		
2- 4	40	850		
2- 5	41	1000		
2- 6	42	1000		
2- 7	43	800		
2- 8	44	900		
2- 9	45	700		
2-10	46	750		
2-11	47	1050		
2-12	48	950		
2-13	49	900		
2-14	50	900		
2-15	51	700		
2-16	52	600		
2-17	53	600		
2-18	54	400		
2-19	55	290		
2-20	56	300		
2-21	57	100		

from the day of operation, averaging 500 cc. per day until the eighth day, when 900 cc. was given. From the tenth through the nineteenth day, 1200 cc. per day was given. After four days at this level, the bleeding stopped. The amount of plasma was decreased to 650 cc. on the twentieth day, and its use was omitted on the twenty-first and twenty-second days. When bleeding from the gastrointestinal tract began again on the twenty-third day, fresh frozen plasma and blood again were administered. At the time of the wound separation on the twenty-sixth day, the volume of fresh frozen plasma was increased to more than 1200 cc. per day for the first six days after its repair. When bleeding was controlled, the intake was decreased to between 750 and 1000 cc. per day. The maximal amount of fresh frozen plasma given was 1800 cc. per day. The record of fresh frozen plasma and blood administered is shown in Table I and Figure 2.

Review of Table I and Figure 2 indicates that 1200 cc. of fresh frozen plasma per day was required to control bleeding in this patient. The clotting time was kept within normal limits when this amount was given. This agrees with the experience of Aggeler,[2] who pointed out that a 60-kg. person with a blood volume of 5000 cc. would require approximately 1500 cc. of fresh plasma given at the rate of about 300 cc. every two hours for the first ten hours to achieve a 30 per cent level of antihemophilic factor. After this initial dose, Aggeler recommended that approximately 1 cc. per kilogram of body weight per hour be given until all evidence of bleeding had disappeared.

Supplying Blood and Plasma

It became apparent soon after the operation had been completed that the patient would require large amounts of blood to replace the loss due to continuous seepage, as well as large amounts of fresh frozen plasma to overcome the deficiency of antihemophilic globulin.

More blood than the six units on hand surely would be needed and most likely in large amounts. An appeal was made to the Milwaukee Blood Center for group O, Rh_1, homozygous rh′ (C) and Fy(a-) bloods. During the illness of this patient, the Milwaukee bank shipped blood to Minneapolis on five different occasions, sending a total of thirty-seven units. These were crossmatched at the Minneapolis War Memorial Blood Bank and twenty-eight of them were used for the patient. In the meantime, the laboratory staff screened a total of 923 group O, Rh positive bloods, of which sixty proved to be compatible. Two units of blood were received from the Mt. Sinai Research Center, Chicago, and one from the John Elliot Blood Bank, in Miami. Fresh frozen plasma was prepared daily by the Minneapolis blood bank and administered at the hospital. During the seventy-one days of hospitalization, the patient used fifty-three units of whole blood, together with 203 units (50 cc.) and 423 units (100 cc.) of fresh frozen plasma. Converted to 500 cc. units of whole blood, the equivalent of 332 units was used.

The patient is now clinically well, having been released from the hospital on February 25, 1956.

This experience emphasized the inadequacy of any one blood bank to meet certain emergencies. The promptness and dispatch with which other community blood banks came to the aid of the Minneapolis War Memorial Blood Bank in this emergency demonstrated how such emergencies can be met and lives saved by co-operative efforts of good community blood banks. The credits for such transactions can be easily cleared by working through the American Association of Blood Banks' clearing houses.

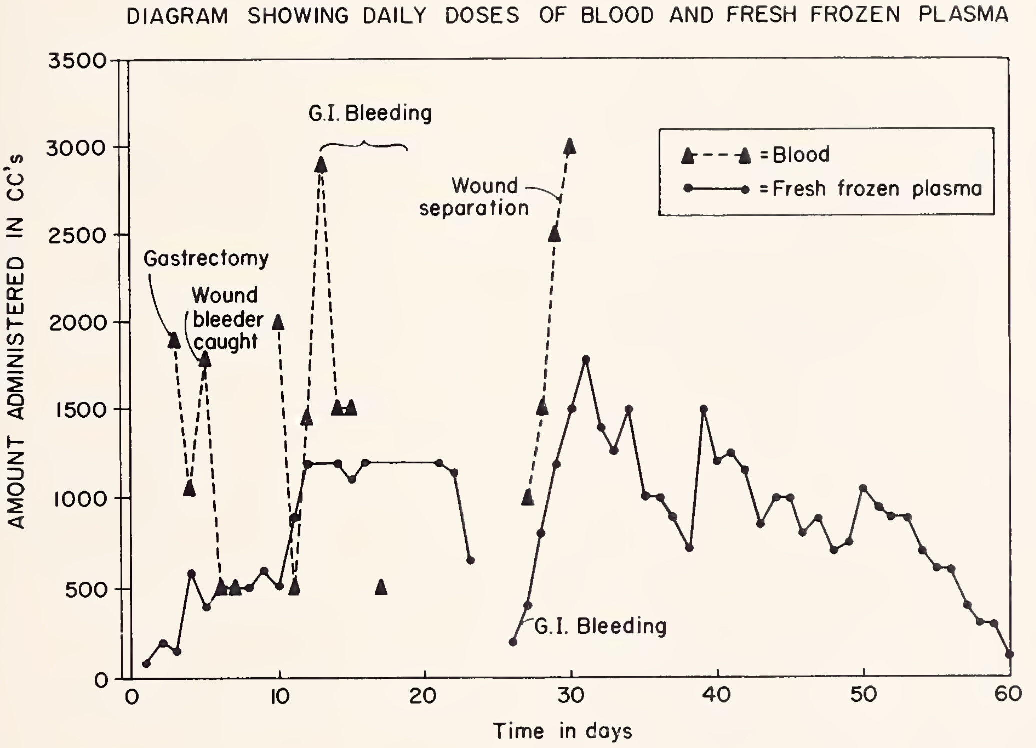

Fig. 2. Administration of plasma and blood to a hemophiliac patient undergoing gastrectomy.

Summary and Conclusion

The role of a number of community blood banks working together in meeting the blood needs in a case of elective subtotal gastric resection, in a hemophiliac patient who was sensitized against rh″(E), hr′(c) and Fy(a+), is described. The importance of community blood banks co-operating in emergencies is emphasized. Working through the American Association of Blood Banks' clearing houses simplifies such transactions.

Acknowledgment

Appreciation is expressed to Dr. Tibor Greenwalt, Milwaukee Blood Center, Inc., who confirmed our antibody findings and sent selected blood to be crossmatched for the patient. Gratitude is also expressed to Dr. Kurt Stern, Mount Sinai Research Center, Chicago, and to Dr. Lloyd Newhauser, John Elliot Blood Bank, Miami, for sending selected blood for this patient.

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Surgical Treatment of Esophageal Hiatus Hernia

A Description of Some Important Details in the Technique of Repair

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THE PROBLEM of hiatus hernia has been appreciated best in recent times. In fact, up until 1921 only forty-four cases of all types of diaphragmatic hernia had been diagnosed during life.[1] In 1926, Akerlund[2] collected sixty cases of hiatus hernia diagnosed during life and reported twenty-four of his own. This reflects the more general use of x-ray in diagnosis. Surgical treatment of hiatus hernia essentially dates from Harrington's report of his repair in sixty cases in 1933.[3] A significant advance in the technique for repair of this hernia was made when a more anatomical closure of the defect posterior to the esophagus was performed.[4,5]

Simple repair of the hernia is sufficient in the majority of cases, but does not always prevent or correct one of the important accompaniments of hiatus hernia, namely, reflux esophagitis with its attendant serious complications. This is apparent from the fact that rather extensive extirpative and reconstructive procedures have been advocated and performed in an effort to correct the complications of reflux esophagitis.[6,7] It is important to note that the changes consequent to esophageal reflux, even though severe, are reversible if the irritant is removed.[8] In the normal person, prevention of reflux has been attributed to actual compression of the esophagus by the right crural fibers as they course around the esophageal hiatus. This arrangement is the so-called diaphragmatic pinchcock[9] which could only function during those times that the diaphragm contracts. At operation, with a finger in the hiatus, a pinchcock effect is difficult to detect. Some observers have described a lower esophageal circular sphincter muscle.[10] This cannot be a constant or a definite structure since other competent observers have been unable to find it in their dissections.[11]

There is interesting experimental evidence that the angle of insertion of the esophagus to the stomach is the important factor in preventing reflux into the esophagus (Fig. 1).[12] The treatment of reflux esophagitis, medical as well as surgical, will have its greatest measure of success if it is based on this experimental evidence.

The indications for surgery in the treatment of hiatus hernia are similar to those for duodenal ulcer, namely, symptoms intractable to medical management or complications of a serious nature such as gastrointestinal bleeding (chronic or acute), esophageal stenosis, gastric ulcer at the neck of the hernia, or perforation of the stomach or esophagus. In some of the author's cases, indication for operation was based on the objective finding of pathology present in other organs, most often the gall bladder.

The symptoms in these cases, however, were characteristic of reflux esophagitis. In weighing the case for surgical treatment in this disorder one notes: (1) that the problem is a mechanical one and might reasonably be expected to respond incompletely to medical therapy, (2) that hiatus hernia increases in size with time, and (3) that Blades[13] reports 45 per cent of his series of sixty-six cases had dangerous complications (bleeding, twenty-two; stricture, eight; perforation of the stomach, one; and carcinoma in abberant mucosa, one). It is generally felt, therefore, that all patients who are acceptable risks, and who, in spite of medical treatment, have a symptomatic hiatus hernia should have surgery recommended to them.

We have had occasion to treat, surgically, twenty-five patients with symptomatic hiatus hernia.* From the symptoms that they present and from the findings at surgery, these patients may be divided into two groups. In the first group (Table I) are those patients who presented excruciating, deep, subxiphoid pain which bores directly through to the back or around both costal margins to the back. This pain appears suddenly,

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Presented as an inaugural thesis before the Minneapolis Surgical Society, November 4, 1957.

*Several of these cases are presented with the cooperation of the St. Louis Park Medical Center, Minneapolis.

TABLE I. (GROUP I) PATIENTS WHOSE PREDOMINANT SYMPTOM WAS SUBXIPHOID PAIN

Pt.	Age	No. GI X-Rays	No. X-Ray Diag. Hiatus Hernia	Diameter Hiatus	Operation	Follow-Up
1. E.R.	57	1	1	5 cm.	H.H. repair	Well 6 yrs.
2. R.C.*	60	1	0	3 cm.	H.H. repair	Recurrence of pain after 1½ yrs. Reexplored. No hernia.
3. L.S.	24	2	0	3 cm.	H.H. repair	Well 3 yrs.
4. J.S.†	65	2	0(fundal mass)	6 cm.	H.H. repair	Well 2 yrs.
5. G.H.	23	2	0	3 cm.	H.H. repair	Well 1 yr. 10 mo. Questionable improvement.
6. P.B.*	34	2	0	4 cm.		Psychiatric care.
7. A.E.	54	1	1	3 cm.		Well 6 mo.

*R.C. and P.B. both had emotional disturbances, the severity of which was not recognized preoperatively. They should not have been selected for operation.

†The fundal mass seen on x-ray proved to be edema of gastric mucosa at site of herniation.

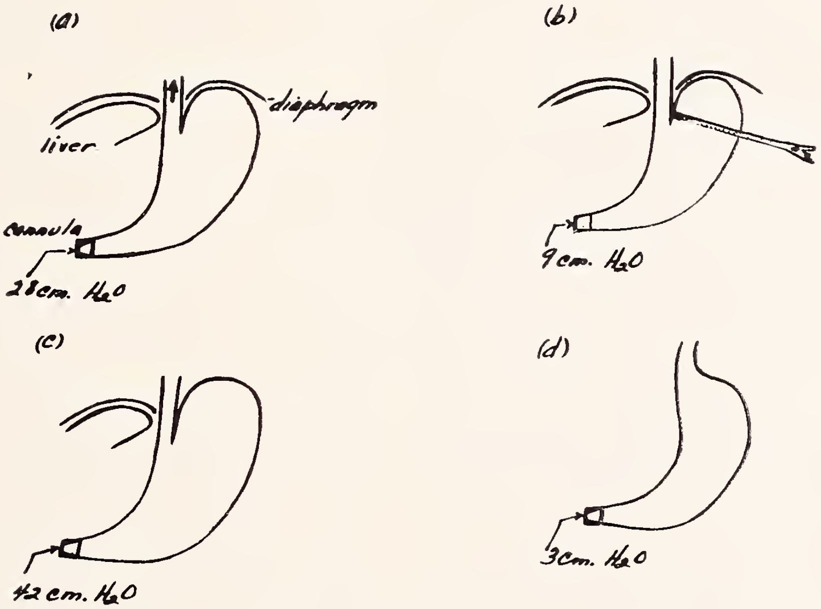

Fig. 1. Illustrates retrograde pressure necessary to overcome resistance of cardio-esophageal junction under varying conditions in young adult male cadavers. (From Marchand) (a) Control. Pyloris cannulated. Stomach undisturbed. (b) Clamp across stomach so as to exclude fundus. (c) Left leaf-diaphragm removed. Abdominal wall open. (d) Esophago-gastric angle abolished by freeing terminal portion and removing liver and right diaphragm.

without warning, persists steadily at maximum intensity for varying periods, and then suddenly and completely disappears. Except for moderate epigastric voluntary muscle-guarding during the attack of pain, the physical examination and all laboratory and x-ray studies are normal. This hernia is extremely difficult for the radiologist to demonstrate. We must, therefore, occasionally rely on the rather characteristic clinical picture to lead us to the diagnosis and to surgery as the only effective treatment. The clinician's failure to appreciate the radiologist's limitation in this area, may lead him to the unjust diagnosis of psychoneurosis or even to the removal of questionably diseased organs as a measure of desperation to bring relief to these patients.

Report of Case

Case 5 in Group I illustrates the foregoing. This young, stable farmer was first struck with severe subxiphoid pain radiating through to the back while sitting in church May, 1951, when he was nineteen years old. The pain lasted twenty minutes and was gone when the physician arrived. He subsequently had many similar attacks, which, as time went on, lasted longer and occurred more frequently. Evidence against this pain being functional in origin is the fact that occasionally as he was being driven to town to receive a hypo for the pain, he would get spontaneous relief, and then would turn around and return home to work. A gall bladder which concentrated well, but emptied slowly, and did not contain stones was removed on January 18, 1954. This did not relieve the attacks. An indication of the frequency of attacks is given by the fact that he received fifty-six hypodermic injections

TABLE II. (GROUP II) PATIENTS WHOSE PREDOMINANT SYMPTOM WAS REGURGITANT ESOPHAGITIS

Pt.	Age	No. GI X-Rays	No. X-Ray Diag. Hiatus Hernia	Diameter Hiatus	Operation	Follow-Up
1. R.B.	28	2	2	6 cm.	H.H. repair	Well 3 yrs. 7 mo.
2. E.H.	63	3	0	3 cm.	G.B. & H.H.	Well 3 yrs. 5 mo
3. R.J.	42	1	1	3 cm.	G.B. & H.H.	Well 3 yrs. 5 mo.
4. F.G.	69	1	0(Ready Regurg.)	4 cm.	Vagotomy Pyloromyotomy H.H. repair	Well 3 yrs. 4 mo.
5. J.N.	63	1	1	6 cm.	H.H. repair	Well 3 yrs. 2 mo.
6. C.B.	55	1	0	4 cm.	G.B. & H.H.	Well 3 yrs. 1 mo.
7. J.G.	64	1	1	8 cm.	H.H. repair	Well 2 yrs.
8. E.H.	59	1	0	6 cm.	H.H. repair C.B.D. stone	Well 1 yr. 10 mo.
9. D.G.	42	2	2	6 cm.	H.H. repair	Well 1 yr. 8 mo.
10. B.J.	38	2	0	3 cm.	G.B. & H.H.	Well 1 yr. 8 mo.
11. E.D.	45	4	1	7 cm.	H.H. repair	Well 1 yr. 6 mo.
12. E.O.	47	1	1	8 cm.	H.H. repair	Well 1 yr. 6 mo.
13. S.H.	65	2	2	10 cm.	H.H. repair	Well 1 yr. 5 mo.
14. H.T.	49	1	0	6 cm.	G.B. & H.H.	Well 1 yr.
15. A.S.	33	2	0	6 cm.	C.D. stone H.H. repair	Well 1 yr.
16. G.C.	62	1	1	10 cm.	G.B. & H.H.	Well 10 mo.
17. E.D.	58	1	1	5 cm.	H.H. repair	Well 9 mo.
18. A.N.	44	1	1	7 cm.	H.H. repair	Well 3 mo.

of either Morphine Sulfate grs. 1/6 or Demerol mgms. 100 from the time of onset in 1951 to December, 1955. At that time, after an extensive study had failed to reveal the diagnosis, an exploratory laparotomy was performed. Careful exploration revealed only a 3 centimeter hiatus hernia and absence of the gall bladder. The hernia was repaired and the patient has been asymptomatic since convalescence from this operation.

Case 3 in Group I presents an almost identical history. This young man had been hospitalized three times because of severe sub-xiphoid pain, and again extensive study did not reveal the diagnosis. He has been asymptomatic since simple closure of a small hiatus hernia defect.

Case 4 is of interest. This sixty-five-year-old man complained of attacks of midline epigastric pain. An x-ray of the stomach was interpreted as showing a mass in the fundus. This was confirmed on a repeat x-ray examination two days later. At laparotomy a hiatus hernia measuring 6 centimeters in transverse diameter was found. A generous gastrotomy was performed and showed only thickening of the stomach wall apparently due to edema from involvement in the hernia. Simple closure of the hernia defect has brought relief.

At operation in these Group I cases, the hiatus is usually found to admit two fingers into a preformed sac. The finding of a preformed peritoneal sac is of prime importance, since by exerting moderate pressure, one can normally insinuate two fingers into the esophageal hiatus. The pain associated with this small hiatus hernia is often sufficient to cause strong, stoical men to pale and perspire and can be best explained on the basis of visceral spasm involving the herniated portion of the stomach. The problem here is the simple mechanical one of herniation through an abdominal wall defect with which we are familiar in the case of inguinal hernia.

The symptoms and findings presented by the patients in the second group, with the possible exception of gastric ulcer, are due to reflux esophagitis. These symptoms and findings include retrosternal burning (heart burn), eructation, dysphagia, vomiting, flatulence, gastrointestinal bleeding, and esophageal stricture. In this group, the diagnosis is made by the radiologist somewhat more easily. We have, however, seen two patients (one of whom is included in this report) where it was possible to establish the diagnosis of hiatus hernia only after four separate gastrointestinal x-ray examinations. In several, it required two x-ray examinations to visualize the hernia. This is not surprising if we consider our experience with inguinal hernia, where we do not expect to demonstrate more than the very largest hernias at every examination. The symptoms of reflux esophagitis are easily elicited and are associated in the majority of cases with hiatus hernia even though such cannot always be demonstrated radiologically. In this group the esophageal hiatus, as examined at operation, tends to be slightly larger and frequently admits three fingers into a preformed sac.

In this second group (Table II), therefore, we have the dual problem of a diaphragmatic defect and incompetence at the esophago-gastric junction. It is felt that these problems are both mechanical. The experimental work referred to above suggests that the acute angle of insertion of the esophagus into the stomach is the factor of importance in maintaining the competence at this union. We have gathered an additional bit of circumstantial evidence to support this experimental study. First, we have found in careful dissection of the esophago-gastric area in autopsy specimens, that in the presence of a significant hiatus hernia, there tends to be effacement of the acute angle of insertion of the esophagus to the stomach as compared with those specimens in which the esophageal hiatus was normal. Secondly, we have found that a real clinical test of the importance of the acute angle of esophago-gastric union in making this junction competent is presented by those patients who have had a resection of the esophagus. In five such patients an attempt was made to restore the normal esophago-gastric angle and re-create the gastric fundus (Fig. 2). This is accomplished by anastomosing the esophagus to the mediastinal side of the thoracic stomach approximately four centimeters caudad from the superior-most portion of the remaining stomach. The cephalad end of the stomach is then sutured in place to the mediastinum above the anastomosis to maintain it in the position of a gastric fundus. Post-operatively these patients have had no clinical symptoms of regurgitation and radiologically the union was competent in spite of the fact that it was now impossible for a "lower esophageal Sphincter" or "a diaphragmatic pinchcock" to function. These patients eat normally and sleep horizontally without fear of regurgitation and aspiration. Aside from the present report, a reconstruction which results in a competent esophago-gastric union would give some meaning to the term "palliative resection" for carcinoma of the esophagus.

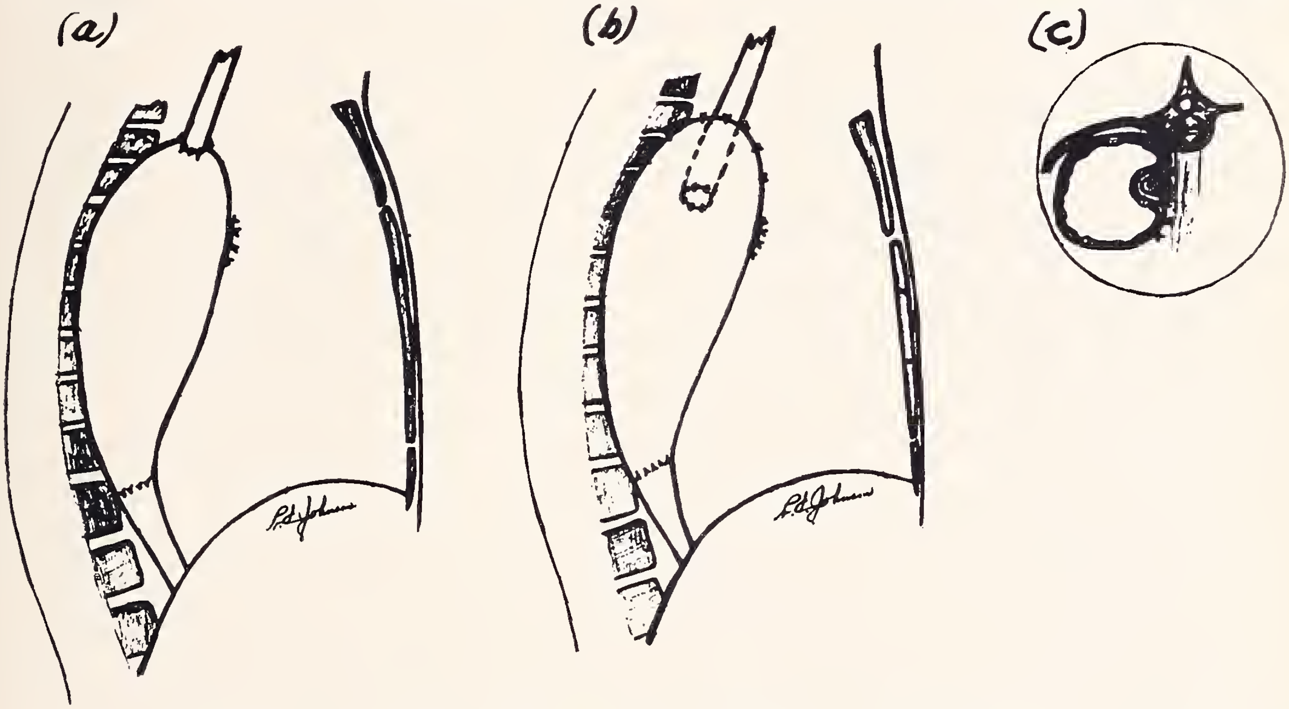

Fig. 2. Restoration of normal esophago-gastric angle and re-creation of the gastric fundus. (a) The type of thoracic esophago-gastrostomy usually pictured. (b) A thoracic esophago-gastrostomy which re-creates a gastric fundus and the acute angle of insertion of the esophagus into the stomach. (c) Horizontal section just above point of entrance of esophagus to stomach.

If we accept from the foregoing the explanation given for the symptoms presented by the Group I and II patients, our surgical objective is clear. In the Group I cases, where there is no symptom of esophago-gastric incompetence, the following procedure aimed at closure of the diaphragmatic defect suffices:

The hernia is reduced by placing traction on the stomach. The peritoneum overlying the esophagus is incised, and this structure with the accompanying vagus nerve is freed up. A penrose drain is passed about it to aid in retracting it inferiorly or from one side to the other as the margins of the hiatus are cleaned and delineated. The margins of the hiatus are then approximated posterior to the esophagus. Generous "bites" are taken on each side of the hiatus. This is especially important on the right where there is considerably less muscle mass in which to place the suture. Here the "bite" should extend well over toward the inferior vena cava, where the tissue is more fibrous and has greater tensile strength. The esophageal hiatus is closed sufficiently so that it will admit in addition to the esophagus and nasal tube, only the tip of the index finger. We have been unable to identify the phrenico-esophageal ligament described by Allison. We do, however, attempt to maintain the esophagus in an abdominal position by incorporating in the most cephalad hiatal suture, the adventitia of the esophagus, posteriorly. Anteriorly, the esophageal adventitia is sutured to the anterior margin of the hiatus. Diaphragmatic contraction places no stress upon this repair, and there is, consequently, no need for right phrenic nerve interruption in these cases (Fig. 3).

In the Group II cases where gastro-esophageal reflux is the primary problem, we must restore the normal acute angle of esophago-gastric union in addition to a simple snug closure of the esophageal hiatus. It is felt that this is best and most surely accomplished by adding to the operation described above, simple suture of the distal esophagus to the adjacent fundus for a distace of approximately three or four centimeters (Fig. 3). The results of this operation have been pleasing to the patient as well as to the surgon (Table II).

Mention should be made of left phrenic nerve paralysis in the surgical treatment of hiatus hernia. This measure is most likely to be effective in those cases with regurgitant esophagitis. There is a rational basis to be found in the work of Marchand and for expecting this procedure to show a degree of success (Fig. 1). We are of the opinion, however, that it is rarely indicated in this or any other condition to interrupt the phrenic nerve and thus defunctionalize one of the most important muscles of the body. This applies particularly in the condition under discussion, where direct attack on the problem is simple, safe, and effective.

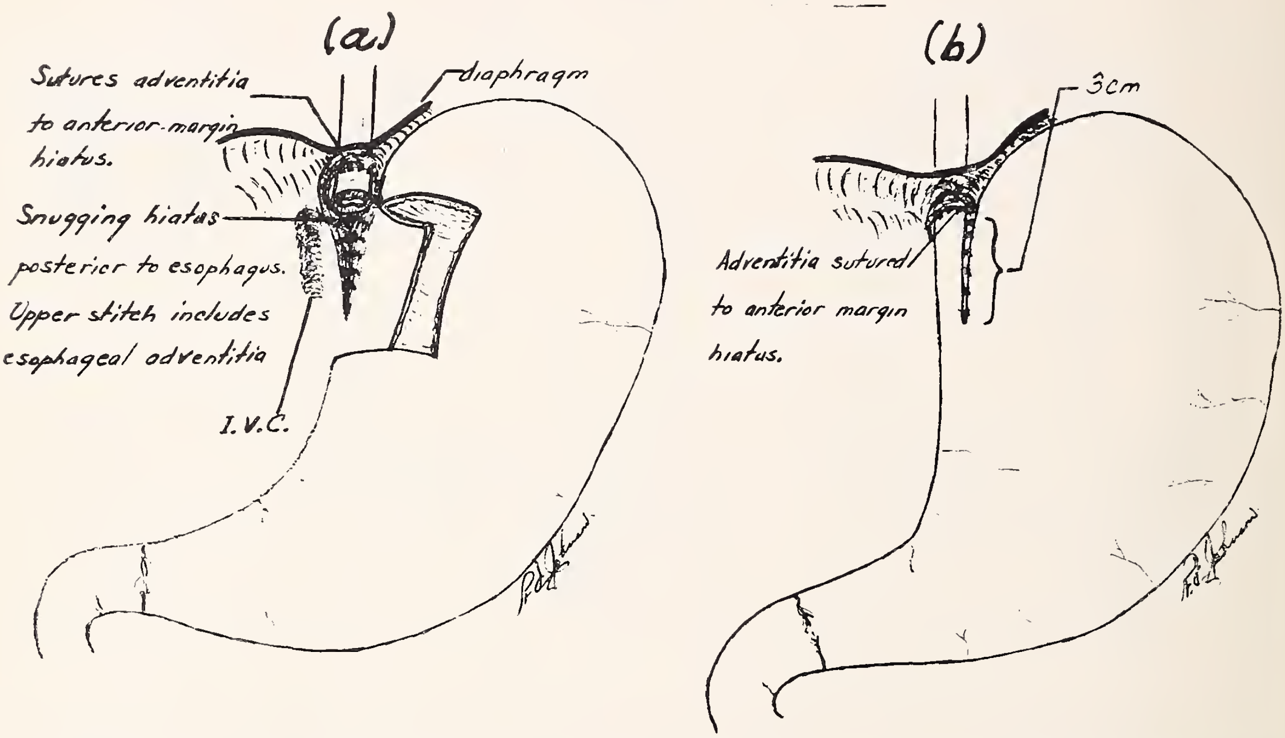
(a)
Sutures adventitia to anterior margin hiatus.
diaphragm
Snugging hiatus posterior to esophagus. Upper stitch includes esophageal adventitia
I.V.C.
(b)
3cm
Adventitia sutured to anterior margin hiatus.

Fig. 3. Restoration of normal acute esophago-gastric angle providing a simple snug closure of the esophageal hiatus. (a) Lower esophagus and adjacent stomach cut away to show closure of hiatus. (b) Lower esophagus sutured to adjacent fundus to restore acute angle of union.

Summary and Conclusions

From a study of twenty-five patients with symptomatic hiatus hernia treated surgically, it is felt that patients with this disorder fall into two groups according to the symptoms they present. In the first group are those who have no regurgitant esophagitis, but are troubled primarily by attacks of severe upper abdominal pain thought to be due to visceral spasm involving the herniated portion of the stomach. In the second group are those patients whose primary difficulty is reflux esophagitis. In both instances the problem is a mechanical one which yields to a relatively simple procedure. The importance of the acute angle of esophago-gastric union in preventing gastro-

(Continued on Page 113)

Peripheral Neuritis

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THE WORD "neuritis" often is used loosely and inaccurately as an elegant variation of the word "pain", particularly when such pain is of undetermined cause. The term "neuritis" is properly and explicitly restricted to designate the more or less prolonged disorders of sensation, motility and visceral function due to the disease or trauma of any peripheral somatic nerve or nerves.

A typical peripheral nerve is composed of motor, sensory and visceromotor fibers. The symptoms produced by the involvement of such a nerve will be due to involvement of the motor or the sensory, or the visceromotor components or various combinations of these three. It is thus possible to have a peripheral neuritis reflected as pure motor symptomatology such as is classically found in the wrist drop in lead poisoning, to have a peripheral neuritis with purely sensory symptomatology such as is frequently found in diabetic neuritis and to have pure visceromotor phenomena such as is encountered in incomplete injuries to the sciatic nerve. Any combination of involvement of the sensory, motor and visceromtor systems may exist and thus the picture presented by a peripheral neuritis may be very complex.

The symptomatology presented will depend upon the type of involvement of the peripheral nerve from which the patient is suffering. Each of the functions will be considered separately here, although, of course, any combination of the three can occur in the same individual.

Types of Involvement

Sensory Involvement.—The subjective symptomatology of which the patient may complain is extremely varied. He may complain of actual pain. However, frequently he complains of numbness and tingling. The patient may complain of paresthesias and finds it difficult to describe accurately the sensations which he is experiencing. Many compare these paresthesias when they involve the feet, for example, to walking on a thick rug. The patient may complain of hyperesthesia or increased perception of sensations. A common type of hyperesthesia encountered is the excessive pain elicited upon stroking the soles of the feet of a patient suffering from alcoholic peripheral neuritis.

The sensory system is composed of essentially two parts: (1) perception of deep sensibility and tactile discrimination and (2) the perception of pain, temperature and light touch. One sensory modality may be involved while the others remain intact. Involvement of pain or light touch can be detected, on careful examination, by using a pin and a wisp of cotton. The use of two test tubes, one filled with warm water and the other with cold water, will detect involvement of temperature. Deep sensation is determined by testing the position sense of the toes or fingers, by compressing tendon between the thumb and forefinger, by using a tuning fork to determine the patient's ability to perceive vibration sense, and by use of a divider with the points being placed at various distances apart and applied simultaneously to the skin. Joint pain is tested by attempting to move the joints through increased ranges of motion. Normally the patient will complain of pain or discomfort when the normal range of motion of the joint is exceeded but as in tabes dorsalis, it may be possible to far surpass this normal range of motion without any complaints on the part of the patient.

In neuritis, there may be great tenderness of the muscle supplied by the injured nerve, which may be sensitive not only to pressure but also to traction. Wartenberg emphasized that in every disease, the peripheral nerves respond with pain to brisk passive stretching and that this is a constant very early and very late sign of neuritis. Chronic dermatitis may occur in the area of cutaneous distribution of an injured nerve.

The velocity of transmission of nerve impulses is related to the caliber of the nerve fibers. On this basis, nerve fibers have been divided into

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three groups: (A) touch, pressure and proprioceptor fibers with a diameter of 1 to 2 microns transmitting a velocity of 30 to 100 meters per second; (B) small myelinated temperature and localized pain fibers transmitting a velocity of 10 meters per second; and (C) unmyelinated diffuse pain and efferent vasomotor fibers transmitted at 1 to 2 meters per second. The rate of transmission averages 27 per cent less in the distal than in the proximal segments indicating that the velocity is closely related to the diameter of the fiber.

Motor Involvement.—The patient usually complains of weakness, clumsiness or inability to do some motor function which he could do previously without difficulty. On objective examination, weakness is demonstrated on testing isolated muscles or muscle groups. Usually the reflex is decreased or absent at the site of the weakness. On observation of the involved musculature, fasciculations may be visible. Partial interruption of a nerve may yield a tonic spasm of muscles which represents an irritative phenomenon.

Involvement of Visceromotor Fibers.—The peripheral nerves carry a varying complement of visceral components. In the median and sciatic nerves, this component is especially large. A burning type of pain is usually suggestive of lesions of these visceral fibers and it may occur independently of impairment of muscle power and cutaneous sensation. Vasomotor and trophic disorders (usually most marked peripherally) include objective warmth or coolness, rubor, pallor or cyanosis, hyperhidrosis or hypohidrosis, edema, atrophy of the skin and subcutaneous tissues, hyperkeratosis, pigmentation or depigmentation, irregular growth of hair and nails, and ulcers. The hair may actually fall out, in complete nerve injuries. With incomplete nerve lesions, there may be an excessive production of hair.

Special Testing of Peripheral Nerve Function

In most instances, by careful neurological examination, it is possible to determine the presence of neuritis. However, in some obscure problems, particularly where the differential diagnosis of hysteria or malingering is entertained, special testing of disturbance of nerve function may be of benefit.

These special methods include:

1. The determination of the electrical reactions of nerve and muscle. By the use of faradic and galvanic stimulation applied to the nerve and to the muscle, the presence or absence of a complete reaction of degeneration or partial reaction of degeneration can be determined.

2. The determination of chronaxie. The minimal strength of current of infinite duration necessary to give contraction is called the rheobasic voltage. The length of time the current twice this strength must flow to stimulate a nerve or muscle is called its chronaxie. Adrian has found that the chronaxie of a human muscle with an intact nerve supply is about 0.00016 second while the chronaxie of the muscle associated with the degenerated nerve is about 0.01 second.

3. Measurement of electrical resistance.

4. The application of paste of iodine and starch to the skin. This is sensitive indicator for the presence of perspiration.

5. The use of the plethysmograph, and the measurement of the local temperature of the skin. The retention or loss of vascular reaction when peripheral nerves have been injured can be inferred by measurement of the local temperature of the skin or by placement of a plethysmograph on the affected extremity. For example, if the median or ulnar nerves are paralyzed, the opposite extremity is plunged into ice water; there results no vascular response in the affected extremity but when the radial nerve alone is paralyzed, the vascular response is normal.

6. Histamine Test. Following the intradermal injection of 0.01 cc. or the application to a fine puncture wound of 1:1000 solution of histamine hydrochloride, normally a flare appears in three to five minutes. The flare is retained in hysterical anesthesia, or anesthesia of central origin. It appears also within the first six to eight days following interruption of a peripheral nerve; after this, however, the flare reaction does not appear in an area rendered anesthetic by a lesion of its nerve supply. As regeneration takes place, the histamine flare returns early.

7. Electromyography.

8. Examination of cerebrospinal fluid.

9. Roentgenograms of the extremities. There may be evidence of pronounced demineralization of bones when the extremity has been deprived of its nerve supply. The epiphyseal lead line may

be of great help in the diagnosis of lead poisoning in children.

Classification and Terminology

Various classifications have been proposed for classifying peripheral neuritis, according to: (1) Its clinical course; that is, acute, subacute or chronic. (2) The nature of the symptoms—referable to motor, mixed or sensory nerves. (3) The distribution of the condition, for example, mononeuritis, mononeuritis multiplex, multiple neuritis, polyneuritis or polyradiculitis. (4) The part of the nerve affected, i.e., radiculitis, ganglionitis, funiculitis, plexitis, neuronitis or peripheral neuritis. (5) The type of tissue affected—parenchymatous or interstitial. (6) Generalized polyneuritis or localized neuritis.

An etiological basis for the classification, however, appears to have the most practical significance and a classification based upon etiology is presented as follows:†

Classification of Neuritis

I. Toxic-Infectious
  1. Guillain-Barre's Syndrome
  2. Exogenous Toxins
    (a) Heavy metals—arsenic, lead, thallium, phosphorus, mercury, manganese, gold, silver, bismuth, copper, antimony (?), zinc (?), and tin (?).
    (b) Organic solvents—alcohol, carbon disulfide, benzene, tetrachlorethane, trichlorethylene, carbon tetrachloride, etc.
    (c) Carbon monoxide.
    (d) Miscellaneous—methyl bromide, triothocresyl phosphate (Jamaica ginger)
  3. Bacterial Toxins—diphtheria, botulinus.
  4. Serum Neuritis—tetanus antitoxin; pneumonia, meningococcus, scarlet fever, and diphtheria sera or antitoxin, antirabic treatment.
  5. Complicating Infections—mumps, scarlet fever, gonorrhea, typhoid fever, typhus fever, chorea, leprosy, measles, rabies, small pox vaccination, infectious hepatitis, and puerperal sepsis.
  6. Drugs
  7. Miscellaneous—insect bites, acrodynia, congenital pink feet, hereditary affliction of the feet, sun burn

II. Traumatic
  1. Fractures and dislocations
  2. Occupational trauma
  3. Trauma in athletics
  4. Trauma during operation or therapeutic procedures
  5. Birth trauma
  6. Trauma in war

III. Vascular
  1. Vascular occlusions due to—arteriosclerosis, mechanical compression (trauma, neoplasm, tourniquets, aneurysm, etc.), periarteritis nodosa, thromboangiitis obliterans, livedo reticularis, embolism, exposure to colds (radial nerve), reaction to radium.
  2. Vascular stasis (Volkmann's ischemia)
  3. Hemorrhage—purpura, hemophelia.

IV. Tumor
  1. Primary—neurofibroma, von Recklinghausen's disease, ganglioneuroma, amputation neuroma, neuromyoarterial glomus, Kaposi's disease, fusiform cysts or ganglions of the nerves, perineural cysts.
  2. Metastatic
  3. Ruptured intervertebral disc.

V. Metabolic
  1. Diabetes
  2. Pernicious anemia
  3. Porphyria
  4. Nutritional—beriberi, pellagra, gastric carcinoma, malfunctioning gastroenteric stoma, obstructive duodenal ulcer, gastric polyps, diaphragmatic hernia, intestinal fistula, prolonged vomiting and malnutrition.
  5. Neuritis of pregnancy.

VI. Hereditary
  1. Peroneal type of muscular atrophy of Charcot-Marie-Tooth (familial)
  2. Hypertrophic neuritis of Dejerine-Sottas (familial)

VII. Miscellaneous—scleroderma, disseminated lupus erythematosis, pregnancy, Boeck's sarcoid, acromegaly, radium, amyloid disease, gout (?).

Summary

1. Peripheral neuritis may present with motor, sensory or visceromotor symptomatology or various combinations of these three.
2. Features of the sensory, motor, and visceromotor involvement in peripheral neuritis are reviewed and the special testing of peripheral nerve function outlined.
3. A practical etiological basis for the classification of peripheral neuritis is presented.

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†The details of all the variations of the involvement in each of these conditions can be obtained by consulting any standard textbook in Neurology.

Use of Primacaine in Intraocular Ophthalmic Surgery

A Clinical Evaluation

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A RELATIVELY new anesthetic agent, 2′-diethylaminoethyl-2-butoxy-3-aminobenzoate hydrochloride (Primacaine hydrochloride) has been studied by a group of investigators[1] and used for various purposes clinically in this country and Canada.[2] The author, under the supervision of Edward P. Burch, M.D.,* has recently conducted a clinical survey of Primacaine in intraocular ophthalmic surgery. No attempt has been made to compare it with xylocaine, procaine, and other anesthetics.

The patients represented a consecutive, unselected, series of intraocular cases operated upon by a group of local ophthalmologists. The patients varied in age from twenty-one to eighty-nine; the majority being fifty years of age or older. The series is comprised of eighty cases of cataract without glaucoma, two cases of cataract with glaucoma, ten cases of glaucoma, two cases of anterior chamber foreign body, and one case of cataract after-membrane. Preoperative sedation consisted of thorazine 25 mgm. or phenergan 50 mgm. with demerol 50 mgm. in patients under seventy years of age. In patients over seventy years of age, the dosage of both drugs was halved.

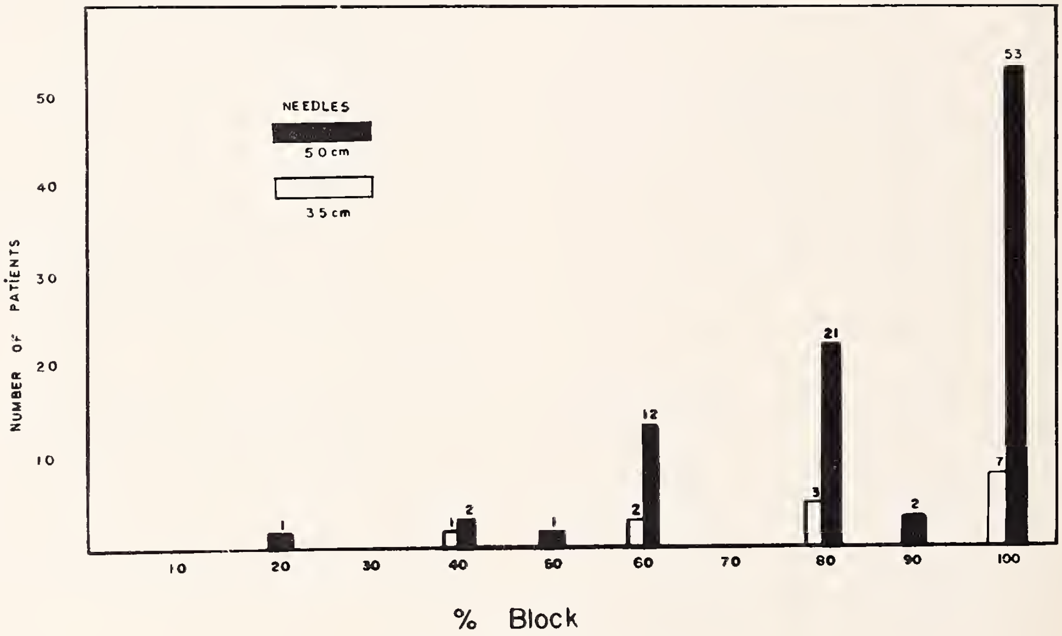

Fig. 1. Ocular akinesia.

The 5.0 cm. 25-gauge needle was used in all but a few cases. In these few cases (most of which were glaucoma) the 3.5 cm. 25-gauge needle was substituted. The trans-cutaneous method of retrobulbar injection and the Van Lint lid block were employed throughout. Primacaine hydrochloride with epinephrine 1:60,000 and hyaluronidase 5 viscosity-reducing units per cc. were used. Two cc. of solution were used for retrobulbar injection and 2.5 cc. for the lid block. The globe was massaged following injection for a period varying from three to five minutes.

The block was evaluated for ocular akinesia, intraocular tension, lid akinesia, and patient comfort during the surgery. Ocular akinesia was evaluated on a percentage basis as advocated by Gifford. In this method 100 per cent indicates complete ocular akinesia, 20 per cent is deducted from 100 per cent for each rectus muscle still active, and 10 per cent for each oblique muscle still active. Intraocular tension was estimated as firm, moderately soft, and very soft. This was evaluated at the time of the corneal section on patients having no previous glaucoma. Firm tension was present when there was a definite tendency for iris prolapse and a lack of corneal wrinkling, moderately soft when there was corneal wrinkling without a tendency for iris prolapse, and very soft when there was corneal indentation. Complete lid akinesia was present when there was an absence of lid squeezing during and following surgery.

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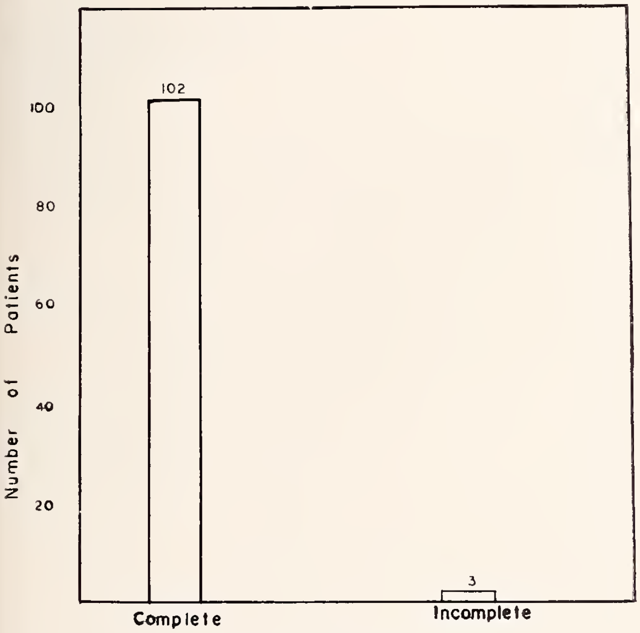

Fig. 2. Lid akinesia.

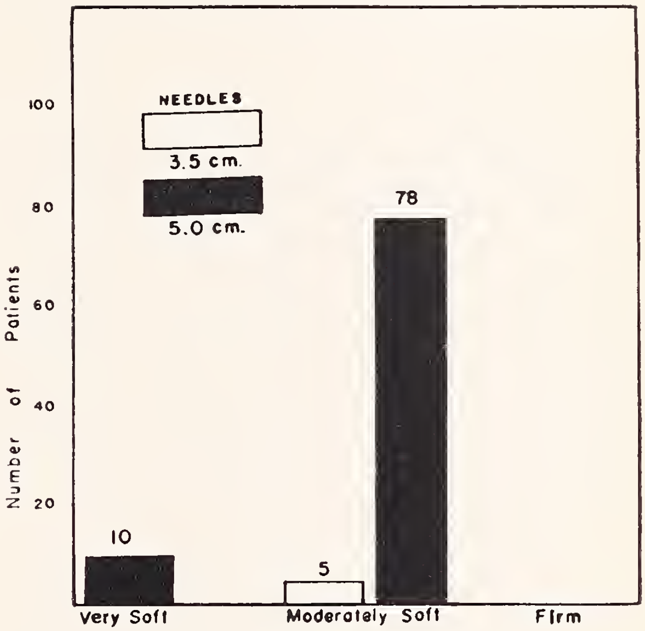

Fig. 3. Intraocular tension.

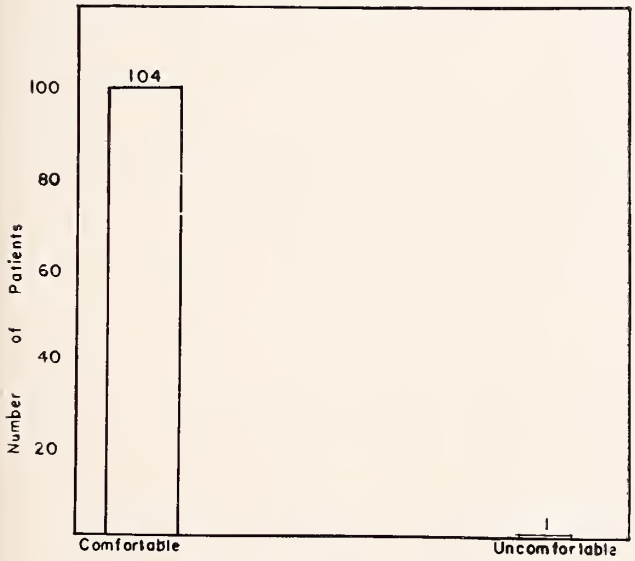

Fig. 4. Patient comfort during surgery.

Conclusion

In conclusion, the clinical results indicate that Primacaine hydrochloride represents a superior anesthetic agent for intraocular ophthalmic surgery.

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Hospital and Hospital Staff Responsibility for Maternal Mortality

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SUBSTANTIAL reductions in maternal mortality rates have occurred within recent years. Gross maternal mortality rates in Minnesota have decreased from 2.03 per 1,000 live births in 1941 to 0.49 per 1,000 live births in 1954, a reduction of 76 per cent. The proportion of preventable deaths decreased in the same period from 73.2 per cent to 30 per cent. In a recent five-year period in Minnesota, only 177 "obstetric" deaths occurred among almost 400,000 live births. A summary of these deaths showed, however, that 45 per cent were considered preventable.[1]

Clearly, room for improvement still exists. The responsibility of patients through failure to avail themselves of care, attempted self-induced abortions, suicides, etc., accounts for a negligible fraction of the deaths. This is in marked contrast to earlier studies such as the "New York Study" which showed that patients were responsible for over one-third of the deaths.[2] It follows that continuing efforts to reduce maternal mortality must be directed primarily to physicians doing obstetrics. This is now being done by education at medical school and at postgraduate levels, at medical society meetings, by publication of maternal mortality study committee findings, and by letters and case summaries directed to the physicians concerned. Since almost all of the deliveries are conducted in hospitals, maternal mortality rates could probably be lowered further by general adoption and enforcement of effective hospital regulations governing the conduct of obstetric practice. Formulation and enforcement of such regulations should be the joint responsibility of the hospital staffs, the hospital administrators and the official health agencies concerned.

The purpose of this paper is to analyze the preventable maternal deaths which occurred in Minnesota during the five-year period 1950-1954, with the aim of determining what improvement could have been made had the hospital practice of obstetrics been better organized.

TABLE I. RESPONSIBILITY FOR PREVENTABLE MATERNAL DEATHS OCCURRING IN MINNESOTA HOSPITALS 1950-1954

Responsible	Number	Per Cent
Physician	66	90.4
Patient	2	2.7
Nurse-anesthetist	2	2.7
Laboratory	2	2.7
Hospital administration	1	1.4
Total	73	99.9

TABLE II. CAUSES OF PREVENTABLE MATERNAL DEATHS OCCURRING IN MINNESOTA HOSPITALS 1950-1954

Cause of Death	Number	Per Cent
Hemorrhage	33	45.2
Toxemia	12	16.4
Infection	8	11.0
Anesthesia	7	9.6
Electrolyte imbalance	3	4.1
Air embolism	2	2.7
Incompatible blood	2	2.7
Heart disease	2	2.7
Diabetic acidosis	1	1.4
Pituitrin shock	1	1.4
Over transfusion	1	1.4
Unknown	1	1.4
Total	73	100.0

During the time period mentioned there were eighty-three maternal deaths in the state which were judged by the Maternal Mortality Committee of the Minnesota State Medical Association to have been preventable. Of these, four resulted from non-obstetric causes and six patients died at home. The remaining seventy-three deaths occurred in hospitals and of these, physicians were judged to have been responsible for sixty-six and the hospitals through nurse-anesthetist, laboratory and/or administrative error were directly responsible for five. The responsibility for two deaths was charged to the patients (Table I). The causes of the seventy-three deaths are indicated in Table II.

From the Minnesota Maternal Mortality Study Committee. Drs. Freeman and Barno are Obstetrician-Consultants for the Committee.

Presented at the meeting of the Minnesota Obstetrical and Gynecological Society, Saint Paul, November 30, 1957.

TABLE III. TABULATION OF DEATHS DUE IN LARGE PART TO INADEQUATE PATIENT STUDY

Cause of Death	Number
Hemorrhage	7
Infection	5
Anesthesia	5
Toxemia	4
Electrolyte imbalance	3
Incompatible blood	2
Heart disease	1
Diabetic acidosis	1
Unknown	1
Total	29*

*39.7 per cent of 73 preventable deaths occurring in hospitals.

TABLE IV. DEATHS DUE DIRECTLY TO FAULTY CONDUCT OF LABOR, DELIVERY AND EARLY PUERPERIUM

Cause of Death	Number
Improper anesthesia	7
Improper use of pituitary extract	7
Accouchement force	6
Inadequate observation of patient in labor and/or postpartum	5
Internal version	3
Inadequate pre delivery preparation	3
Total	31*

*42.5 per cent of 73 preventable deaths occurring in hospitals.

Fifty-four of the deaths occurred in out-state hospitals and nineteen in the four largest cities in the state. This distribution of the deaths is out of proportion to the distribution of births occurring in the state, these being divided approximately equally between the four largest cities taken together and the remainder of the state. Data previously published showed that the "physician responsibility maternal mortality rate" was two and one half times higher in rural than in metropolitan areas.[1] Obviously, some fundamental differences in the hospital practice of obstetrics exist in the two areas.

Factors which have been evaluated as to their influence on the occurrence of these seventy-three preventable deaths, include patient work-up (history, physical examination, pelvic mensuration, laboratory and x-ray studies), conduct of labor and delivery (including administration of anesthesia), and consultation. In many instances more than one of these factors were involved in a death. This overlap accounts for the fact that in Tables III, IV and VI, percentages total more than 100.

Patient Work-up

There were many deficiencies in the most basic patient work-up and observation. The effects of such deficiencies are partially shown in Table III. In twenty-nine instances, the entire hospital record consisted of scanty nurses' notes only, while another nineteen did not have a complete history and physical examination recorded. Only two anesthesia records were found in a series of seven anesthetic deaths. Consultation records and progress notes by the physicians were almost uniformly missing or deficient. In not a single case was the prenatal record made a part of the hospital chart. Pelvic measurements were almost never recorded.

TABLE V. CONSULTATIONS OBTAINED IN CASES OF PREVENTABLE MATERNAL DEATHS IN MINNESOTA HOSPITALS 1950-1954

	Number	Per Cent
None	37	50.7
Inadequate and/or delayed	30	41.1
Adequate	6	8.2
Total	73	100.0

TABLE VI. TABULATION OF CASES OF MATERNAL DEATHS IN WHICH LACK OF CONSULTATION PLAYED A MAJOR ROLE

Cause of Death	Number
Hemorrhage	27
Toxemia	5
Infection	5
Electrolyte imbalance	3
Heart disease	2
Anesthesia	2
Diabetes	1
Unknown	1
Total	46*

*63 per cent of 73 preventable deaths occurring in hospitals.

Some of the disasters resulting from the lack of simple studies are illustrated by the following cases. Four patients who ultimately died of the effects of anesthesia did so because pelvic measurements were inadequately evaluated. Two of these occurred at the time of cesarean sections which were done because of "outlet contraction" in one patient and breech presentation in the other. Neither simple clinical nor x-ray pelvimetry was done in these cases. The other two developed prolonged obstructed labors due to unrecognized pelvic contractions and died of the effects of general anesthesia and aspiration in association with difficult operative deliveries. A fifth patient whose pelvis was never adequately evaluated died of a ruptured uterus following internal version and breech extraction done because of questionable obstruction of labor.

A patient died of the effects of a ruptured uterus after attempted internal version and breech extraction for obstructed labor. The obstruction was due to the presence of a pelvic tumor which was undetected because pelvic examination had never been done.

Another patient was hospitalized for fifteen days at four months gestation with abdominal pain, nausea and vomiting and other signs of an acute abdomen. Four days after discharge she was readmitted and she expired twelve hours later. Autopsy showed a ruptured appendiceal abscess. On neither admission was a history or physical examination recorded. No laboratory work was done except for one hemogram and a single urinalysis, and the single consultation obtained was delayed and inadequate.

Still another patient was hospitalized for a total of three weeks. She had been vomiting for six weeks. No history, physical examination or laboratory work was done and no consultation was obtained. The patient died of shock, dehydration and electrolyte imbalance at fifteen weeks gestation. Another patient known before pregnancy to have congenital heart disease was admitted for a period of four days with signs and symptoms typical of congestive failure. No history or physical was done, no x-ray studies, electrocardiagrams or consultation was obtained and no treatment directed at the congestive failure was given. The patient was readmitted four days later still in failure and she died seventeen hours after admission.

Lack of adequate laboratory and x-ray facilities or failure to obtain minimal laboratory studies when available, together with laboratory errors, were major factors in twelve deaths. Some examples included three patients who died of unrecognized electrolyte imbalance, none of whom had any pertinent laboratory work done. A known diabetic who died in diabetic coma after more than three days of hospitalization had no laboratory work done except for a single urinalysis. Another patient died of peritonitis and septicemia five days after classical cesarean section for obstructed labor. The hospital had no facilities whatever for bacteriological study. In one of the deaths due to obstructed labor, difficult midforceps extraction, general anesthesia and aspiration (previously referred to), the hospital x-ray department had predicted that there would be no dystocia. This conclusion was arrived at from study of a single simple flat film of the pelvis with no provision made for correcting for distortion. Two patients died from administration of incompatible blood as a result of laboratory typing errors.

Conduct of Labor and Delivery

Forty-two and one-half per cent of the deaths being considered could be directly attributed to faulty conduct of labor, delivery and early puerperium including improper administration of anesthesia (Table IV). Sixteen or almost one-half of all the deaths from hemorrhage resulted directly from procedures which would never be allowed in a well regulated obstetrical department. These included six who died of hemorrhage from lacerations following manual dilatation of the cervix, three from ruptured uteri caused by performance of unindicated internal version and extraction and seven who died when recognized safety rules regarding the use of pituitary extract were violated.

Five patients in the hemorrhage group died almost directly as the result of inadequate observation in labor and/or the postpartum period. The following example is illustrative of this group. The patient was delivered by an unindicated version, performed because of questionable obstruction. The pelvic capacity, however, had never been adequately evaluated. No blood pressures were taken during labor, during the operative procedure or postpartum. One hour postpartum, simple observation of the patient made it obvious that she was in shock. One pint of blood was given but the shock was never corrected and the patient died four hours postpartum.

Three deaths from hemorrhage occurred principally because of lack of preparation in advance, when hemorrhage could have been anticipated. In numerous other instances, this was a less direct cause. One of these patients was hospitalized for two weeks with pain, anemia, a history of a missed menstrual period and bloody vaginal discharge. The pre-operative diagnosis was never established and a laparotomy was done without intravenous fluids being started or any blood being crossmatched. The patient went into shock during surgery at which time an ectopic pregnancy was found. She died one hour later before any effective anti-shock therapy could be given. Another patient died in a large metropolitan hospital of hemorrhage at cesarean section. No blood had been crossmatched pre-operatively. Still another died of pastpartum hemorrhage due to uterine atony five hours after delivery. No blood was made ready beforehand although the patient was bleeding excessively on hospital admission and a total of only 500 cc. of blood was given.

Proper supervision of anesthesia departments would probably have avoided all seven anesthetic deaths. Four of these resulted from spinal anesthesia, all as a consequence of administration of an overdose of the anesthetic agent. One patient died as a result of violation of safety rules regarding the administration of trichlorethylene and two others died of aspiration after being given general anesthesia in circumstances where this was obviously dangerous. Both patients had been fed and had vomited prior to administration of the anesthetic.

Consultation

Adequate consultation was obtained in only six cases, delayed and/or inadequate consultation in thirty and none at all in thirty-seven (Table V). The unavailability of consultation or, more often, the failure of physicians to call available consultation when indicated, constituted a major factor in no less than forty-six deaths (Table VI). Many examples can be given. A multipara known to have been delivered previously by classical cesarean section was admitted with complaints of abdominal pain and vaginal bleeding. She was in shock on admission with a blood pressure of 70/40. Fetal heart tones disappeared shortly after admission. Diagnoses considered were placenta praevia, appendicitis and placental abruption. The patient expired in shock after minimal anti-shock therapy, seven and one-half hours after hospital admission. Autopsy showed a ruptured cesarean section scar.

In another case, an inadequately qualified consultant was called to see a patient after she had been in hard second stage labor for four hours with no progress being made. Signs of uterine rupture appeared shortly following this. The patient was given subcutaneous pitocin and a difficult forceps extraction of a 6000 gram fetus was done. The patient expired shortly thereafter of shock proven at autopsy to have been due to undiagnosed ruptured uterus. Another patient was admitted at thirty-one-weeks gestation with severe hypertension, four plus albuminuria and epigastric pain, the significance of which was not understood. An unqualified consultant who also failed to appreciate the significance of the epigastric pain, advocated membrane rupture although the cervix was firm and closed. The patient developed convulsions and died undelivered three and one-half days after admission. Still another patient had her uterus incompletely emptied following incomplete abortion. The uterus was packed. Blood replacement was deficient and antibiotic therapy was hopelessly inadequate. She developed signs of peritonitis and probable septicemia and died after six days of hospitalization with no consultation having been obtained. Numerous other examples illustrating the critical effect of inadequate consultations could be cited.

Comment

It is evident that the hospitals and their organized staffs were in too many cases remiss in failing to insist upon adequate patient work-up including history, physical examination, basic laboratory and x-ray data and incorporation of prenatal records including pelvic measurements. Routine observation of patients during labor and delivery and in the early postpartum period was often deficient. The hospitals, for the most part, have failed to define and enforce adequate rules and regulations regarding the performance of unindicated operative deliveries and the improper use of oxytocics. Inadequate organization and supervision of the administration of anesthesia was a large factor. A simple rule limiting the maximum dose of an anesthetic agent to be used for spinal anesthesia in pregnancy is clearly indicated for all hospitals. Anesthetic records were seldom kept. Equipment for administration of oxygen and for aspiration was often not available. None of the patients was observed postpartum in "a recovery room" and trained personnel in general are not available for administration of obstetric anesthesia.

Failure to insist upon adequate consultation for such things as obstetric operations, prolonged labors, toxemia, hemorrhage, retention of the placenta and puerperal sepsis played a major role in 63 per cent of the deaths. The ready availability of consultation in the large cities is manifestly a factor in the great disparity in the maternal death rates between the rural and metropolitan areas of the state. Making available adequate consultation might be considered a worthwhile project for the State Health Department or the Minnesota Obstetrical and Gynecological Society. However, in addition to increasing the availability of consultation, it is clear that a fundamental change in attitude is required on the part of many physicians toward the calling of consultation. Frequently, adequate consultation was available but was not called for fear of "loss of face" or because of pride, ignorance or overconfidence on the part of the physician. Adoption by all hospitals of a regulation making consultations mandatory for such things as operative deliveries, prolonged labors, all forms of obstetric hemorrhage or toxemia, and so forth, could make a very real contribution.

It seems obvious that one of the major factors in the disparity of maternal mortality rates between metropolitan and rural areas in the State of Minnesota is the difference that exists in the organization within hospitals and in the staff control of important features of their conduct of obstetrics. With a few exceptions, all of the metropolitan hospitals are departmentalized and adequately organized. The conduct of obstetrics is closely supervised. Consultations are made readily available and, more important perhaps, are required under certain definite conditions. Hospital regulations forbid such outmoded procedures as accouchement forcé. They clearly define safe methods of using pituitary extracts, require adequate patient workups, have better organized anesthesia departments, and so forth. The few metropolitan hospitals which have failed to conform to these standards have also been the largest contributors of preventable deaths in those areas. Of nineteen deaths occurring in the four largest cities in the state eight occurred in two hospitals.

Summary

A remarkable 76 per cent drop in maternal mortality rates was achieved in Minnesota between 1941 and 1954. This commendable improvement is probably the result of a number of factors such as improved undergraduate training of physicians, increasing postgraduate and refresher courses in obstetrics, widespread use of chemotherapeutic agents, and early and adequate blood replacement. It includes an increasing percentage of babies born in new, modernized, and better staffed hospitals, from 49 per cent in 1935 to 99.1 per cent in 1955. In addition, public health measures such as education of the public to the importance of early and adequate prenatal and obstetrical care, as well as maternal mortality surveys for the education of physicians, are other factors in the reduction of maternal mortality. Perhaps the greatest credit is due the physicians in the state, most of them general practitioners, who are actually doing the obstetrics.

Despite the great achievements of the recent past, it has been shown by the Maternal Mortality Committee's studies that room for improvement still exists. It is with this in mind that an analysis of seventy-three preventable maternal deaths occurring in hospitals in the State of Minnesota in the five year period of 1950-1954 is presented. An attempt is made to determine what improvement could have been made had the hospital practice of obstetrics been more adequately controlled. Forty-two and one-half per cent of the deaths could be directly attributed to faults in the conduct of labor, delivery and the early puerperium which should not be tolerated in well organized hospitals.

Patient work-up including such simple things as history, physical examination and basic laboratory and x-ray studies was deficient in the majority of cases. This failure was considered a definite contributing factor in 39.7 per cent of the total deaths. Adequate consultation was obtained in only 8 per cent of the cases and lack of such consultation was a major factor in 63 per cent of the deaths. It is concluded that in spite of continuing improvement, further strides can be made in reducing maternal mortality by properly organizing and supervising the hospital practice of obstetrics. The time has come for each hospital accepting obstetric patients to give this serious consideration.

Formulation of a detailed workable set of recommendations for the organization and regulation of obstetrical departments in all hospitals in the state should be undertaken by the State Health Department in co-operation with representatives of other interested groups. Such groups should include the state and local health departments, state and local medical societies, the state hospital association and state and local obstetrical societies.

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Discussion

A. B. Rosenfield, M.D.—In the discussion of the five-year summary of maternal mortality in Minnesota, presented by Doctors Freeman and Barno, I would like to consider first, the present situation; second, what has been done to reduce maternal mortality; and finally what additional steps are necessary to further reduce mortality to the so-called irreducible minimum. It should be emphasized that great progress has been made in the field of obstetrical care. Due credit must be given to the general practitioners who provide most of the obstetrical care in Minnesota as well as to the obstetricians for the excellent record they have established. In 1941 when the Minnesota Maternal Mortality Study first began, the rate was 2.03 per 1,000 live births; in 1954, it was 0.49 per one thousand, a reduction of 76 per cent. During this same period the proportion of preventable deaths decreased from 73.2 per cent to 30 per cent, a 60 per cent reduction. During the five-year period of the study, 1950-54, almost 400,000 live births occurred with 177 maternal ("obstetrical") deaths, a rate of 0.44 per one thousand live births. This was among the lowest rates in the United States. Excellent as this record was, the important fact is that seventy-three deaths or 41 per cent were considered preventable by the committee and that the maternal mortality rate was more than two and one-half times higher in the rural than in metropolitan areas.

A brief résumé of the factors responsible for the preventable deaths previously discussed by Dr. Freeman includes:

Deficiencies in patient-work-up and observation as evidenced by inadequate medical records, inadequate evaluation of pelvic measurements, and failure to obtain laboratory studies.

Faulty conduct of labor and delivery.

Lack of adequate consultation.

Failure to define and enforce adequate rules and regulations regarding operative deliveries and use of oxytocics.

Lack of resuscitative equipment and unqualified anesthesia personnel.

Lack of adequate laboratory or x-ray services and facilities.

What has been done to reduce maternal mortality? The State Department of Health is legally responsible for the issuance of licenses to operate hospitals, sanatoriums, nursing homes and boarding care homes under certain rules and regulations. An advisory board of nine members makes recommendations to the State Board of Health and assists in the establishment of rules, regulations and standards. Included on this board are four members appointed by the Minnesota Hospital Association and two physicians appointed by the Minnesota State Medical Association.

The governing body of the hospital or the board of trustees is responsible for its management, control and operation. It appoints a hospital administrator and the medical staff and formulates the administrative policies of the hospital. The governing body is responsible for the physical plant, equipment, facilities, and the employment of personnel. The medical staff is responsible to the governing body of the hospital for the clinical and scientific work of the hospital and for advice regarding professional problems and policies. This is a part of the statutory regulations for the licensing and operation of hospitals. The enforcement of hospital regulations should therefore be the joint responsibility of the hospital board of trustees, the hospital administrator, the hospital medical staff and the State Health Department.

Metropolitan hospitals are departmentalized or completely organized. The obstetrical service is closely supervised. Consultations are readily available and are usually required under definite conditions. Outmoded procedures are prohibited. Adequate patient work-ups are required. There are adequate laboratory and X-ray services and well organized anesthesia departments. This, no doubt, explains why only 25 per cent of the preventable deaths occurred in the metropolitan areas.

During the past decade, there has been improved undergraduate training of physicians, increasing postgraduate and refresher courses in obstetrics for physicians and nurses, refresher courses for paramedical personnel, widespread use of chemotherapeutic agents, and availability of blood for replacement. Public health measures have included maternal mortality studies for the education of physicians and hospitals, as well as education of the public to the importance of early and adequate prenatal and obstetrical care and where it may be obtained.

In the out-state areas many hospitals have in the past been entirely inadequate in size, equipment, facilities and budget. During the past ten years the Hill-Burton federal hospital construction program has made possible the construction of forty-nine general hospitals with 2,237 beds in the rural area. In addition, approximately 2,000 more beds have been provided in modern facilities entirely at local expense. As a result, there has been marked improvement in facilities and equipment which has reduced the overcrowding.

In addition to the physical plant, however, another serious problem which must be met, is the provision for adequately trained hospital personnel. Such personnel are in short supply and are especially difficult to recruit for small out-state hospitals. To help meet this problem, the State Department of Health has been engaged in recruitment activities and regional refresher training courses since 1950. However, since the first of January, 1957, it has been possible to intensify efforts through a U. S. Public Health Service grant. The objective of this demonstration is to improve patient care services in Minnesota hospitals by raising the level of practice of paramedical personnel. Three avenues of approach are used: (1) recruitment of new people into the various health fields, (2) training of existing personnel, and (3) recruitment of inactive trained persons. This is being attempted in the fields of medical technology, anesthesia (nursing), dietetics, medical records, and physical and occupational therapy. The Study employs a staff of consultants in these fields who are visiting hospitals to assemble data to be used as a baseline. On the basis of these findings, there will be determined ways and methods to improve care being given to patients in Minnesota hospitals.

And now, what additional steps are necessary to further reduce maternal mortality? As a result of visits to about one-half of the hospitals in the State, the Hospital Services Demonstration Project makes the following recommendations:

Medical Records.—Every hospital should have a well organized medical staff. The staff and hospital administrator should be record-minded. The staff should appoint a medical records committee which meets regularly and has authority to promote good standards and to reject substandard records. There should be a rule regarding delinquent records. A qualified record librarian is essential for satisfactory medical records. It is important to note that the major responsibility for an adequate medical record rests with the attending physician and he assumes final responsibility for its completion and accuracy even though the task may have been performed by an intern or resident.

The medical staff should determine what constitutes a complete and satisfactory record rather than have each staff member make a personal decision. An adequate patient work-up should include the medical and obstetric history, physical examination, and basic laboratory x-ray studies. The Joint Commission on Accreditation of Hospitals requires a prenatal history and physical examination on all obstetrical patients. The physician's prenatal record including pelvic mensuration should, therefore, be incorporated into the hospital record.

The medical staff should establish rules and regulations defining the types of cases in which consultations are mandatory and should require that consultation reports include findings and recommendations and be written and signed by the consultant. According to the Standards for Hospital Accreditation, consultations are required in all cases of major surgery in which the patient is not a good surgical risk, in cases of obscure diagnosis or questionable treatment, in all first cesarean sections, sterilizations and curettages.

Recognizing that consultations are frequently difficult to obtain in out-state areas the State Department of Health is attempting to employ a well-trained and qualified obstetrician on a full-time basis to provide such consultations to physicians on request. If this is not possible, other approaches will be necessary. However, as Doctors Freeman and Barno have pointed out, the mere availability of consultation is not sufficient. There must be a willingness on the part of physicians to make use of consultation services as they become available. This is well illustrated by the autopsy service for maternal deaths available through the State Health Department where such local service is not readily available. During the past three years exactly three requests have been received for autopsies.

Anesthesia.—Every hospital should have qualified anesthetists. With the limited supply of medical anesthesiologists who are found almost entirely in the metropolitan areas, trained nurse anesthetists are necessary. Six small hospitals visited to date use chloroform for obstetrics, six have no gas machines or resuscitative equipment. The registered nurse on the obstetrical floor of many small hospitals gives the anesthetic, usually with no previous training and scanty current instruction. Frequently, the attending physician sets the regulators on the gas machine and the nurse holds the mask. This apparently meets hospital medical staff approval. There should be training centers set up for giving basic instructions to registered nurses who must give anesthesia to obstetrical patients or in emergencies. The use of regional and pudendal block by the physician would require only analgesia for delivery.

Every hospital should have modern anesthesia and resuscitation equipment and trained personnel to use it, including a relief anesthetist. Training in anesthesia is necessary in small hospitals. Staff conferences should include the nurse anesthetist so that she may feel herself to be a part of the surgical team. There should be regulations governing the giving of food or fluids to women in labor. The provision of adequate anesthesia equipment and trained staff is the final responsibility of the governing board; but the regulation of maximum dosages of an anesthetic agent for spinal anesthesia is the responsibility of the hospital staff, the staff physicians.

Qualification.—Medical technologists and technicians should be qualified. In-service training has been conducted for office, clinic and hospital technicians. Obstetrical patients should be typed early in pregnancy and if Rh negative they should be checked periodically for sensitization. If not possible locally, Rh titers can be done at most regional hospitals, at the Minneapolis War Memorial Blood Bank or at commercial laboratories. A copy of the patient's work-up done in the physician's office should be sent to the hospital when the patient is admitted. At least, the physician should notify the hospital laboratory of a possible Rh problem. When an emergency crossmatch is ordered, careful typing is necessary as well as a check for ABO incompatibility and possible Rh sensitization. Plasma should be available in an emergency when a quick crossmatch cannot be done. It can be stored at room temperature for one and one-half years. In case of drastic emergency when whole blood is necessary, low titered O negative blood may be given without crossmatching.

Summary

Let me quickly summarize the points I am trying to make: There are statutory hospital rules, regulations and standards, even though minimal and general. There are standards for hospital accreditation. There are specialized consultants and services available from the State Health Department. Studies of hospital facilities and services are in progress and recommendations for improvement are being developed. In-service training of medical and paramedical hospital staff is available. But, utilization of avaliable resources depends on hospital management—the governing body of the hospital and its administrator; and even more so, on the hospital medical staff. The State Health Department has legal authority to enforce regulations and standards. But the legal approach, while immediate, has no carry-over value. We, therefore, prefer to use the educational approach. It takes longer and is more difficult but it has more permanent carry-over values and thus is more desirable in the long run.

Our most effective public health programs have been the result of the joint cooperative effort of all agencies,

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False Albuminuria Due to Para-aminosalicylic Acid

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SINCE 1946, with the addition of streptomycin to the therapeutic regimen, the prognosis of the tuberculous patient has steadily improved. This has been due almost entirely to long term chemotherapy combined with selective resection. It is now common for patients to continue taking this medication on a prescribed program a year or two after discharge from a sanatorium, not because the disease was particularly advanced or recalcitrant but because the overall results are definitely better.

PAS (para-aminosalicylic acid) is used in nearly all instances in combination with streptomycin, or isonicotinic acid hydrazide or both. PAS is taken orally in the dosage of about 12 gms. daily and is absorbed rapidly from the gastrointestinal tract. After a single dose of 4 gms. of the acid form, the maximum blood concentrations range from 3 to 12 mg./100 ml. within ninety to one hundred twenty minutes. The blood level rapidly falls within three to five hours. Most of the PAS is excreted in the urine; usually, between 80 to 85 per cent can be recovered in the urine within ten hours of administration. The rapid excretion results in high urinary concentrations (400-900 mg. per 100 c.c.) of the various forms —free and conjugated amines.

Because, in our judgment, many patients who have had superior results from the modern antituberculous therapy have an excellent prognosis, it was decided to accept the application for insurance of a young doctor who had been discharged from a tuberculosis sanatorium less than a year previously and who was still on anti-tuberculous chemotherapy (INH and PAS). When his urine was examined at our laboratory, it was found to contain 75 mg. of "albumin" per 100 c.c. of urine by the sulfosalicylic acid method when compared with the Kingsbury-Clarke Standards. Two additional samples verified the "albuminuria." This, however, could not be verified by the doctor applicant. It was then decided to mail a urine sample without adding our preservative tablet. This proved to be the key. Subsequently, urine samples from ten patients who were taking PAS at Glen Lake Sanatorium near Minneapolis were tested for albumin. When all were found to be negative, our preservative tablet was added to each specimen and allowed to stand overnight and again tested for albumin. All were positive for "albumin" in concentrations varying from 5 to 100 mg. per 100 cubic centimeters. Eight of the ten patients were also taking INH and two were taking streptomycin in addition to the PAS.

The preservative tablet used by our laboratory is made by Magar Chemicals Inc., and contains "benzoic acid, a trace of a mercuric compound (one mg. per tablet) and the gradual evolution of formaldehyde in amounts too small to affect the chemical tests."

It was then decided to ask the co-operation of Merck, Sharp & Dohme. Investigation by their Chemical Control Division confirmed the false albumin test. Although they did not determine the full composition of the preservative tablet nor investigate the exact mechanism of the interference they did establish that Sodium PAS plus benzoic acid does not give false albumin tests, whereas Sodium PAS plus formaldehyde yields an interference in the form of turbidity. Formaldehyde thus appears to play a role, but it was felt that the complete mechanism is more complex and might involve the mercuric compound as well. Since formaldehyde is not present in the preservative tablet *per se* but is gradually evolved, this would account for the delayed interference.

Because of the increasing number of people

(Continued on Page 120)

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Delivered before the Medical Section of the American Life Convention, Lake Placid, New York, June 1, 1957, and to the Minnesota Trudeau Medical Society meeting at the Minnesota State Sanatorium, Ah-gwah-ching, August 10, 1957.

Case Presentation

Colorado Tick Fever

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IN THESE days of widespread travel and rapid means of transportation, physicians are likely to encounter illnesses that originate in areas other than their own. Because these illnesses are not ordinarily encountered the diagnosis may be unnecessarily delayed. Colorado tick fever is just such an illness which is easily suspected clinically if one is aware of its existence, but to the unfamiliar it poses a difficult problem. An instance of Colorado tick fever was recently encountered which is being reported to alert other physicians to its existence. This illness is unusual in this section of the country and only recently was it included for the first time in a standard textbook of medicine.[1]

Case Report

J. S., a thirty-seven-year-old white man, a physician. vacationed in Scenic Park, Minnesota from June 2 through June 5, 1957. During this time ten to fifteen ticks were removed from his clothing, but to his knowledge he was not bitten. On June 7 he left Minnesota for Colorado, and on June 9 he parked his trailer house in the Rocky Mountain National Park in Colorado. During the day of June 10 the patient hiked about the mountains of this area. Approximately five ticks were removed from his clothing during the hike, and at about 10 P.M. that same day a partially embedded tick was removed from his abdomen without difficulty.

At about 9 P.M. on June 13, almost exactly three days after he observed the tick bite, malaise, chilliness and slight headache were noted. These symptoms rapidly increased in severity and within one hour a fever of 102° was noted along with shaking chills and a severe frontal and retrobulbar headache. The patient continued febrile until the afternoon of June 16 (about three days). At this time no symptoms other than some lassitude and easy fatigue were noted. The patient was first examined on June 15, at which time no abnormalities other than fever were noted. A blood count at that time disclosed a total white blood count of 3,200 with 54 per cent neutrophils, 34 per cent lymphocytes and 12 per cent monocytes.

The patient continued with some malaise and lassitude until about 6 p.m. on June 18, at which time chilliness and headache recurred. Within four hours these symptoms reached a severity similar to the original episode of June 13. The patient's fever remained almost constantly at about 102° until it returned to normal early in the morning of June 21. A blood count done on June 21 disclosed a white blood count of 1,600 with 41 per cent neutrophils, 49 per cent lymphocytes, and 10 per cent monocytes. This degree of leukopenia occasioned some alarm on the part of the physicians who were caring for the patient, and because of this he was admitted to the Worthington Municipal Hospital on June 21, 1957 for more complete evaluation.

Shortly after admission to the hospital the patient's clinical course was reviewed and a diagnosis of Colorado tick fever was suggested. The patient was afebrile throughout his four-day stay in the hospital. White blood counts obtained on June 23 and 24 were 2,200 and 3,250 respectively. Other laboratory data included absent antibodies for tularemia and brucellosis, and absent heterophile antibodies. Blood cultures were also negative. A blood sample obtained from the patient on the afternoon of June 21 was mailed to the Rocky Mountain Laboratory in Hamilton, Montana. From this specimen a pathogenic agent was isolated by the inoculation of suckling mice which was identified as Colorado tick fever. A rise in the neutralizing antibody titer for Colorado tick fever virus was demonstrated in subsequent blood samples.

For a period of four days following the last episode of fever, the patient experienced marked lassitude and was easily fatigued. He returned to work on June 26 but it was not until about June 30 that he felt entirely normal. A white blood count on July 22, 1957 was 5,350 with 59 per cent neutrophils, 32 per cent lymphocytes, 5 per cent eosinophils and 4 per cent monocytes.

Discussion

The diagnosis of the above case was suggested by the typical clinical course of Colorado tick fever. After an incubation period of four to six days the illness begins quite suddenly with chills, fever, headache, general aches and anorexia. These symptoms continue for about two days and are followed by a period of remission lasting several days. This period is followed by a relapse of the original symptoms which usually lasts for several days and is at times more severe and of longer

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From the Worthington Clinic.

duration than the initial episode. At times variations are seen. Third relapses are known and a continuous single episode of fever lasting five to seven days is at times seen. Physical examination is not revealing except for occasional mild erythema of the skin and conjunctival injection. Clinically, Colorado tick fever is indistinguishable from dengue except for the occurrence of a rash in the latter illness.

The most important laboratory lead to the correct diagnosis is the constant leukopenia which is usually most marked during the second episode of fever. Counts of less than 2,000 are common. Definitive diagnosis can be made by the isolation of the specific virus from the blood of the patient or the demonstration of the development of antibodies. The virus is isolated by injecting suspected blood intraperitoneally into three- to four-day-old mice.[2] For this purpose, mailed blood specimens are satisfactory, and the virus has been isolated from blood obtained anytime within the period of fever.

Thus far Dermacentor andersoni has been the only tick definitely shown to be the vector for this illness.[2] However, there are some reports of the virus isolated from ticks as far distant from the natural habitat of Dermacentor andersoni as Long Island, New York. To date, all instances of the illness have originated in the specific region to which Dermacentor andersoni is confined. The case presented no doubt originated in Colorado since Dermacentor andersoni is not found in Minnesota and the interval of onset after exposure to ticks in Minnesota was in excess of the known incubation of Colorado tick fever.

Summary

A case of Colorado tick fever has been presented which was recognized by a typical diphasic febrile course and leukopenia. The diagnosis was confirmed by the isolation of the specific virus from the blood of the patient.

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SURGICAL TREATMENT OF ESOPHAGEAL HIATUS HERNIA

(Continued from Page 98)

esophageal reflux is emphasized by the result in five cases of esophageal resection where an attempt was made to restore this angle. In occasional instances where radiologic demonstrations of a hiatus hernia is difficult, it may be necessary to rely on the history to lead us to the correct diagnosis and proper treatment.

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Continuation Study

Expected Behavior in Children

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Des Moines, Iowa

CERTAIN patterns of behavior occur with such regularity at different age periods in infants and children that they may be regarded as normal or expected. Yet such patterns of behavior are frequently of concern to parents and lead to parent-child conflicts which could be avoided with better understanding. The specific items selected for discussion are those which experience has shown to be most frequently bothersome to parents.

No two children are exactly alike. Even identical twins have some distinguishing features. On the other hand, it is equally true, as has been shown by Gesell and others, that within certain individual variations, all normal children pass through much the same maturational sequences in growth and development from birth to adult life. It is customary to classify growth and development under four broad categories—physical, intellectual, emotional and social. The maturational sequences of physical and intellectual growth are readily understood by everyone. Thus the child sits before he stands, stands before he walks and walks before he runs, and he progresses in height and weight from year to year. Likewise, he begins his intellectual development with his first smile at around six weeks of age, reaches for objects in his vicinity by five or six months, says a few words shortly after a year of age, talks pretty well by two and begins to read in the first grade of school. To expect any different sequences of events in these categories of growth and development would, even to parents, be ridiculous. But the behavior patterns of emotional and social development, while equally characteristic in the different age periods are less obvious and therefore less readily accepted. For instance, it is a little difficult for parents to be sanguine about such behavior as temper tantrums, negativism and curiosity about how the opposite sex is constructed—especially when the latter leads to investigation with the neighbor's child. When the four-year-old calls his mother "an old dummy" or tells her to "shut up," it takes a good deal of persuasion to convince her to ignore such language on the basis that "that's the way four-year-olds behave, and it's but a passing phase."

Physicians whose practices embrace the care of children have an opportunity to add greatly to the day-to-day satisfaction and pleasure parents have in living with their children by anticipating for them and interpreting to them expected patterns of behavior as the child proceeds from one age period to another. It is to be hoped, too, that such an approach might lessen the chances of later development of serious behavior problems which will require the services of the psychiatrist to straighten out. It has been well said that most of the problems parents have with their children arise from the fact that children behave as children. Too often parents expect adult behavior at an age when it is impossible of achievement, and they feel thwarted and incompetent by their failures.

Let us consider some of the expected types of behavior in the first two years of life, which is a most important period for the establishment of good parent-child relationships, and let us begin with the crying infant. Why do infants cry? What is meant by "three months colic?" Among the many suggested causes are swallowed air; allergy to milk, orange juice, or fish oil; hunger; excessive fat or carbohydrate in the formula; too small hole in the nipple in bottle-fed infants; enterospasm; "imbalance of the autonomic nervous system"; lack of parental affection; and a host of others. Brennemann, in about 1935, stated that "colic" is due to an "excessive accumulation of gas in the intestines fostered by the highly fermentable content of breast milk" and that, as a result, "the over-distended intestines become kinked and acutely obstructed at the bends." Attempts to overcome this obstruction, he stated, give rise to colic in the same manner as pain is produced by obstruction in a ureter or in a bile duct. It is doubtful

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if many today would agree with this view of Brennemann's.

On the other hand, "full of gas" and "gas on the stomach" are common complaints voiced by parents about this period of infancy. That its source is largely swallowed air can be demonstrated very simply. An x-ray film ten to twenty minutes after birth will show the stomach already distended with air. Two hours later the air will have progressed through most of the small intestine and by six or seven hours will have reached the rectum. Thus attempts to rid the baby of gas are bound to be futile, since obviously there is no such thing as a "gas-less" baby.

Aldrich studied infantile colic as one of his first projects when he went to the Mayo Clinic in charge of the Community Child Health Research Project. Fifty babies in the newborn nursery were observed day and night for eight days. Observations were kept on the total number of crying episodes, the duration of each crying period, and the time of day when the crying occurred. As nearly as possible the cause of each crying episode was recorded. An average of eleven crying episodes per baby occurred per twenty-four hours. The longest period was four hours, and the shortest three-quarters of an hour, with an average of two hours per baby per day. The peaks of crying came at 6:00 P.M. and at 12:00 midnight when there was a minimum of nursing service available. In a subsequent study, Aldrich reported that increasing the personnel at these periods reduced the crying by fifty per cent. Thirty-five per cent of the babies cried because they were hungry, 10 per cent because they had soiled diapers, and 20 per cent because their diapers were wet. In 35 per cent no apparent cause for the crying could be ascribed. Aldrich speculated that the changed environment from intra-uterine to extra-uterine existence played a part. In the uterus was darkness, quiet, and a soothing rocking motion as the mother moved about. Externally there was noise, light, clothing, changing temperature, and lack of rocking.

Most practitioners would probably believe that babies really start to cry after they go home. In a study in homes using mothers as observers, however, Aldrich found there were only four crying episodes per day on the average, as compared to the eleven in the hospital. One inference to be drawn from these studies is that crying is directly related to the amount of attention received by the baby.

Whatever the cause of crying, or "three months colic," it is an extremely disturbing factor to parents, particularly to those with their first-born child. What is to be done about it other than frequent formula changing or giving a sedative drug such as phenobarbital? Several possibilities must be mentioned. First of all, one must understand that crying in the infant in the first few months of life is a protective reflex designed by nature to call attention to the infant's needs at a time when it lacks other means of making its wants known. It is a disappearing reflex, for, as cortical development takes over and the baby has other means of expressing its needs, the crying reflex gradually disappears. This protective reflex is an extremely wise provision on the part of nature. One wonders what the chances would have been for survival of the human race without it. Crying in the young infant, then, may be thought of as expected charactristic behavior for this period, just as there are other characteristic patterns of behavior in other periods of childhood.

Time should be taken by the physician sometime during the mother's pregnancy preferably in parents' classes or during the postpartum hospital period, to educate the parents to expect that the baby will cry when they go home. It is generally accepted, at least among the laity, that subsequent babies are more easily managed than first babies. There is, of course, no difference in the babies, but it is the parents who have changed. Having been through the experience once, they no longer become panicky every time the baby cries. Crying and "colic" are seldom troublesome factors in the home when there is an experienced and competent baby nurse in charge or when the baby is in the hospital attended by skilled nurses. Certainly, any baby that cries excessively should be comforted. An intelligent search for the cause should be made. It would be tragic to overlook an incarcerated inguinal hernia, an intussusception, or an abscessed ear in a crying infant on the assumption that its crying was due to some vague disturbance called "colic." If hungry, the baby should be fed, even though it may not be the scheduled time. If the whole feeding is gulped down it should be given more and the formula subsequently strengthened if need be.

When there is a reasonable chance that the crying may be due to milk allergy, the baby should be given a milk substitute on a trial basis. In general, however, frequent changing of formulas is unlikely to improve the situation. Certainly serious thought should be given before taking a thriving baby off the breast merely because it is crying.

By the end of three months most parents have become accustomed to the task of raising a baby, and the period of crying or "three months colic" has come to an end. The remainder of the first year is usually rather peaceful and pleasant. Only a few minor things arise to disturb the equanimity of the parents. One of these is thumb sucking, which is of such common occurrence in infants that is may be classed as a normal behavior trait. "But won't it deform the baby's jaws?" is the parental complaint. They can be assured that if the practice stops by four or five years of age, as it practically always does simply because the child has matured sufficiently to put infantile methods of behavior behind him, the jaws will in the next few years of development resume their normal contours. A comparable example is seen in the spontaneous straightening of the legs by six or seven years of age when they were bowed or knock-kneed at two. The psychological effect or yanking the infant's thumb out of his mouth can be brought home rather sharply by suggesting to the mother that she yank the cigarette out of the mouth of her unsuspecting husband every time he starts to smoke. A little time out may well be taken to explain to parents that the habit children have of putting everything into their mouths may at one time have served a useful purpose. It is conceivable that in the ancient struggle for existence life itself may have depended upon the chance that what was put in the mouth might have some nutritional value. Such an explanation as this seems preferable to a Freudian concept of sexual gratification.

Somewhere around six months of age, most infants will exhibit a fear of strangers. Mother is likely to be apologetic for her infant's behavior, and explain that she has not had him out among people very much. Again, she needs to be reassured that such behavior is quite normal. In fact, it is a useful index of normal intelligence, for mentally deficient children are unable to differentiate between familiar features and those of strangers. By ten or twelve months of age most infants will have matured sufficiently no longer to be afraid of strangers unless advances are made. If one is imaginative, it is not too difficult to speculate that "fear of strangers" is a protective reaction that at one time served a useful purpose. When danger threatened in the appearance of something unfamiliar, such as a roaming forest animal in search of lunch, the lusty howl of the threatened infant quickly brought father with his stone axe to the rescue.

About the end of the first year infants develop the habit of body curiosity. They poke their fingers into their ears and navels, and girls into their vaginas, while boys handle their genitals. Sometimes girls sit with one finger poked into the vagina while twisting their hair with the other hand or sucking a thumb. Mother does not mind the ear-fingering very much, although practically all mothers at one time or another request that the ears be examined. Nor does she object particularly to explorations of the navel with the examining finger, but she can no longer restrain herself when the curiosity extends to the genitals. It may or may not help to point out to her that so far as the baby is concerned there is no difference between the genitals and the ear. The sex idea is in the adult mind, not in the infant's. The stage of body curiosity will pass whether mother does anything about it or not, but it is best to ignore the whole thing.

When the second year approaches, a new set of circumstances arises which makes the relative tranquility of the first year seem mild indeed. Now cortical development has proceeded to the point where the infant has ideas of his own. Moreover, he has developed the motor ability to enable him to attempt to put his ideas into effect. He has now become a personality in the family whose behavior and actions have to be reckoned with. Whether the family is to live in comparative peace or turmoil depends upon how situations are handled. The second year is one of the most, if not the most, important of all the formative years. The physician may well spend half an hour sometime between the tenth and twelfth month with parents, apprising them of what lies ahead.

The conversation goes something like this: "You've had your easiest year. You could put the baby in his bed or in his play pen and he'd stay there. Feeding hasn't been much of a problem. But in the year ahead of you, you're going to be confronted with an entirely different situation. It is a year when the baby will be forming many fundamental health or behavior attitudes. You'd like them to be good ones so you can live with him pleasantly, but whether they are good or bad depends in large measure upon how you manage him. I should like to have you think of yourself as a teacher as well as a mother in your approach to the tasks that lie before you. The school teacher achieves her goals of teaching children to read, write, and spell by pleasantly conducted daily lessons. She doesn't expect the child to learn to read the first time a book is presented to him. Nor does she do the reading for the child. Instead, she helps him as much as is necessary, which is quite a bit at first, but with gradual shifting over to the child as his capabilities increase until finally he goes pretty much on his own. Instead of book-learning you are going to teach behavior attitudes, but you go at them in the same way. Now what are these behavior attitudes with which you'll be concerned during this next year? Briefly, they are eating, sleeping, play, temper-tantrums, training. Let me tell you a little about each of them."

Sometime around the end of the first year it may be expected that most children will slow up in their intake of food. The reason for this is that the rapid growth of the first year has come to an end. Average weight gains for the first year of life are approximately fourteen to fifteen pounds. During the second year the gain drops to around five or six pounds. Obviously, less food is required when weight gain is slow than when it is rapid. Parents who are unaware of these facts become concerned about the decreased food intake and frequently resort to various subterfuges to persuade the child to eat, not what he wants, but what they want. Not infrequently this marks the beginning of an eating hassle between parents and child which may last throughout childhood until the rapid growth spurt of puberty is reached, and the child again consumes large quantities of food. Most of this unpleasantness could be avoided by some timely advice from the physician in advance. When parents are expecting the decrease in food consumption, they can accept it calmly. They should be told that under no circumstances are they to urge, coax, or force food. It should merely be presented to the child and if he turns his head away, the spoon should quietly be placed back in the dish. If after a few more trials it becomes clear the child is not interested, the meal is terminated pleasantly and with no show of emotion. To eat when one is hungry is a pleasant experience, but to be forced to eat when one is not hungry is unpleasant, and in children usually leads to conditioning against the spoon and the whole performance of being fed.

It should be stressed, too, that most infants somewhere around a year of age make their first attempts to feed themselves. These attempts should be encouraged, not discouraged by keeping the food out of the child's reach, or by tying up his hands so the mother can have an unimpeded pathway to his mouth. Food ends up in the child's hair and is spilled on the floor, but some of it gets to his mouth. With a little experience and help from his mother, he will improve, and before long he will graduate to a spoon because it works better. Many children are capable of feeding themselves completely by thirteen or fourteen months of age, and all of them by eighteen months, provided the mother does a proper job of teaching. Mothers who have not been informed about the importance of teaching children to feed themselves at an age when they are capable of doing so are likely to insist upon doing the feeding themselves because it is less messy, and they can get through the task more quickly. Once children have acquired the ability to feed themselves, they should be expected to do so from then on. No eating problem can arise so long as the parents refrain from interfering. Some years ago, approximately 35 per cent of the preschool children brought for consultation came with the complaint "My child won't eat." What was really meant was "My child won't eat what *I* want him to." Today, eating is seldom a problem among the children whose parents have been educated in advance. Parents should remember that children are naturally dawdlers and should not expect them to attack their food with the gusto of a farm hand. It should be remembered also that many children do not want or require the three meals a day characteristic of adult eating. Many eat heartily only once a day, others may eat small amounts for two or three days, then eat several heavy meals, and some may eat lightly for a week or so only to make up for it in the next two or three days. Whatever the pattern of eating, it is interesting to observe that children who are permitted to run their own eating affairs maintain their positions on their growth charts with surprising constancy, despite the parents' protestations that "he doesn't eat a thing."

Eating is one of the commonest problems parents have with their children. In normal children, in most instances, it is an unnecessary cause of parent-child conflict.

Sleep disturbances are another common problem. Some children have never learned to sleep well at night, dating perhaps from the period of three month's colic when a sleep resistance habit became rather firmly entrenched. There are many others, however, who have slept well throughout the first year but who at about one year of age raise a fuss about going to sleep. Frequently this coincides with the child's newly acquired ability to pull himself to his feet. Whereas formerly, when put to bed, he was content to lie quietly he now pulls himself to his feet, shakes the side of the bed vigorously and demands to be taken back into the family circle. How the parents meet this situation the first time may have much to do with the sleeping pattern for many months. If the child is picked up, rocked, fed or put in bed with the parents, a habit is established and it may be expected that the performance will be repeated nightly, and perhaps many times throughout the night. One harassed mother had been up with her two-year-old six and eight times every night for a full year, and the habit had started under the exact circumstances described above.

The answer is not to be found in sleeping bags, restraining jackets, or tying the child down in bed, but rather in teaching him that once he is put to bed it is expected that he will stay there during the night. The mother should, of course, go to him at once, first, to make sure that he is not in trouble and second, to prevent fears or anxiety about darkness or being left alone. He should be left in his bed, however, with firm instructions to lie down and go to sleep, accompanied by a motherly pet on the head or even a hug and a kiss. Then the mother leaves. If the commotion continues, she returns in ten or fifteen minutes and repeats her admonitions, but perhaps in a little firmer tone. This may have to be repeated several times, but the mother should emerge the victor. Managed in this way, the usual outcome is that the child, after a few nights, abandons the struggle and acquires an acceptable sleeping habit. Here again is illustrated the importance of teaching the desired behavior rather than resorting to punishings or sedative drugs.

A third pattern of expected behavior about which parents may well be given some advance information is the tendency most children have, after they have acquired the ability to walk, to get into things. In their explorations they flit from object to object pausing only briefly to familiarize themselves with something new. They climb anything climbable and handle anything available. They cannot be expected to know that things will break, that they may be expensive, or that they are dangerous. Adults should know that the child in this period has the whole world of things and of people before him about which he has to learn.

What may one do with him during his waking hours now that he has outgrown his play pen? There are two choices: let him have the freedom of the house, or assign him a limited area such as a room with a gate at the door. Some parents take the position that they are going to have things in their home just as they were before the baby came and that they are going to teach him to leave things alone. This seems unwise. Perhaps it can be done, but if so there will need to be such constant conflict between the child and mother that her "no" eventually is likely to convey little meaning because it has been used so often. If the child is to be given free run of the house, the least that might be done to lessen conflict is to remove temptation by putting out of his reach those things which he is not to handle.

A far better arrangement is providing the child a place to play downstairs, such as a room with a gate at the door, where everything is his to do with as he wishes. Parent-child conflict is avoided, mother can go about her work in peace, and yet the open door provides an opportunity for social contact between them. An additional advantage is that this arrangement reduces the risks of accidents, both from trauma and from poisons. It is to be hoped that the physician in his interview at this age period will stress to the mother the necessity of checking her household to make sure that hazards are reduced to the minimum.

Children who are taught from the beginning to be contented in a restricted and safe play area, particularly in the forenoon when mother has work to do, make life much more pleasant for both mother and child. In the afternoon the child is entitled to a tour of exploration about the house or out of doors so that his education will not be neglected.

Temper-tantrums also have their beginning around the first year of life and are to be expected for a couple of years thereafter. Children and adults have the same emotional set-up, but it takes a greater stimulus to call forth an explosion of temper in an adult. The infant has a temper-tantrum over a minor stimulus such as inability to accomplish something he is trying to do or upon being restrained from carrying out some project he has in mind. The treatment is not to throw a bucket of cold water on him, to spank him, to shut him up in a closet, or even to put him to bed, all of which add to his emotional upset. Rather should the storm be ignored, or if the task he has been attempting to perform has proved too difficult, a friendly helping hand may be proffered. The outcome to be avoided is the continuation of temper-tantrums into an age period when they normally should have ceased, because the child has found that his behavior is an effective method of gaining a desired goal. Calmness and aloofness until the storm is over should be the mother's attitude, to be followed by a hug and a kiss. Thus she indicates her love for him as her child, but her disapproval of his behavior.

Finally, a few words about toilet training. Again, there are two ways of going about it. One is the reflex catching of stools and/or urine beginning at any age after the child is able to sit well unsupported. It should be noted that this is not training. True training is not possible until the child has matured sufficiently to understand the sensation of needing to empty his bladder or bowels, seeks a repository, and is capable of releasing urine or feces into it. Most children are eighteen months to two-and-a-half or even three years of age before this degree of maturity is reached. It is best to postpone training attempts until this older age period is reached.

When training is begun, the approach should be not to try to catch every urination on the hour or every stool at exactly one-half hour after breakfast, but rather to teach the child how people use toilet seats. It may seem to the adult that anyone ought to know enough to go to the toilet, but it must be remembered that infants do not; they have to learn the technique. Usually a nursery chair is preferable to a "toidy" seat on the adult stool. If the latter is used, a stepladder should be provided so the child can climb up by himself. The child should be directed to go to the toilet seat as near to the time as possible when a bowel movement or urination is to be expected. He should then be told to get his clothes off and as much assistance as necessary should be provided. He should get onto the toilet seat by simself and be left on for a period of a few minutes. Whether he performs or not, he is shown how to wipe himself and how to flush the toilet if the adult toilet is being used. He should be allowed to get off the toilet seat by himself and assisted in putting his clothes back on. Contrast this with the usual procedure when the mother carries the child to the toilet, lays him down on the floor or bed, takes off his pants herself, places him on the toilet, wipes him herself, lifts him off, puts his clothing back on, and then lets him go. Mother is beautifully trained, but the child has no responsibility whatever. Many children at two years of age can go to the toilet entirely by themselves. There is, however, no particular objection to mothers placing their infants on the toilet seat at an earlier age in an attempt to "catch" the bowel movements, if they wish to do so. Often this succeeds very well, but there should be no illusions about calling it training, for it is not. It is merely "catching" and should be abandoned at the first sign of objection on the part of the infant. The latter is equally true when objections are encountered during training attempts at the older age period. Conditioning against the toilet seat, which may delay training many months, is a real risk when attempts are made too frequently or when over-anxious or unpleasantness is exhibited by the mother.

The child may emerge at the end of the two-year period feeding himself, sleeping throughout the night, reasonably happy in his daytime play, not using temper-tantrums excessively as an attention-getting device, and perhaps with some degree of success in use of the toilet. If so, living together should be a pleasant experience for both child and parents. Furthermore, the basis for the continued development of healthy mental attitudes should have been soundly laid.

In the subsequent years of childhood there are other characteristic patterns of behavior which will not be discussed in detail here. Advance warning at the appropriate age period concerning these should be an assumed responsibility of the physician in his care of the well child, however. A few illustrations may be cited. Between two and three years of age most children show an interest in wanting to dress and undress themselves. Assisted, the task can be accomplished to a surprising degree by the age of three or a little after. Practically all children can be taught to take their own baths by two and a half to three years of age. Fears manifest themselves between the ages of two and four, and vivid imagination crops up during the same period. "Sassiness" can be expected between four and six. Children try out expressions they have heard such as "you old dummy," "I hate you," and "Shut up." Ignored, the use of such language soon dies out. When reacted to violently, however, as commonly occurs among parents, children tend to continue its use because it has been found effective.

One final illustration should be mentioned because of its possible serious consequences. This is the occurrence of body curiosity in the opposite sex which may be expected to appear in many children around the age of five or six. Children of the opposite sex inspect one anothers' genitals out of curiosity to note how they differ. Such behavior is no indication that these children are on the road to becoming sex perverts in later years, but by the violent reactions of some parents it is obvious that they think so. Clearly, the sex idea is more in the adult mind than in the child's. Magnification of the incident out of all proportion to its significance runs the risk of leaving a lasting impression on the child's mind. Parents should be forewarned to meet the acute situation calmly but not to ignore it completely. What is called for is instruction in social customs which decree that we keep our clothes on in public but do as we please in the privacy of our own homes.

Summary

Within reasonable variations most normal children pass through patterns of behavior at different age periods which can be regarded as expected. Physicians can render a helpful service to parents by anticipating for them and interpreting to them these expected patterns of behavior. The objective is to secure more daily satisfaction for parents in living with their children and to prevent later more serious behavior problems.

Expected patterns of behavior selected for discussion in the first year of life include infant crying or "colic," thumb sucking, fear of strangers, and body curiosity.

The importance of the second year of life in the establishment of desirable behavior attitudes has been stressed. Eating, sleeping, play, temper-tantrums, and toilet training have been considered in detail.

Additional expected behavior in later age periods has been mentioned briefly. Body curiosity in the opposite sex is a not infrequent form of normal behavior among five and six year olds. Its management to avoid lasting psychic trauma to the child has been emphasized.

RESPONSIBILITY FOR MATERNAL MORTALITY

(Continued from Page 110)

organizations and groups interested in the problem. We therefore welcome the suggestion by Drs. Freeman and Barno that a detailed workable set of recommendations for the organization, regulation and conduct of obstetrical departments in Minnesota hospitals be developed by representatives of state and local medical societies, state and local obstetrical societies and the state hospital association, in cooperation with the Minnesota Department of Health. We request the active cooperation and support of the Minnesota Obstetrical and Gynecological Society first, in developing and establishing effective recommendations for the proper hospital care of obstetrical patients; and second, and even more important, in stimulating hospital medical staffs to accept and carry out such recommendations. This approach should help reduce and ultimately to eliminate all possible preventable factors in maternal mortality.

FALSE ALBUMINURIA

(Continued from Page 111)

who are completing their treatment after discharge from a sanatorium by continuing their drug regimen even after returning to work, a false-positive test for albumin produced by the presence of PAS and certain preservatives in the urine is of sufficient importance to warrant the present report.

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Clinical-Pathological Conference

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CASE PRESENTATION—MINNEAPOLIS VA HOSPITAL

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This was the first and only admission of this patient to our hospital. He was a fifty-eight-year-old white married fuel oil salesman from St. Paul, who had complained of severe malaise with afternoon fevers and night sweats for three months. Just before the onset of these symptoms he had noted increasing fatigability and he decided to take his family with him on a vacation trip to Mexico. During the trip he developed a "cold" and after arrival experienced sweats and a fever as high as 102 degrees F. He was attended by a Mexican physician who made a diagnosis of pneumonia and gave him penicillin injections. While in Mexico the patient and members of his family experienced diarrhea. Soon after his return home he noted increasing weakness, anorexia, weight loss, afternoon temperature elevations and drenching night sweats.

Ten weeks before admission to the VA Hospital he entered a St. Paul hospital. At that time he appeared acutely ill and a definite hepatosplenomegaly was noted. No lymphadenopathy could be detected. Laboratory tests revealed a hemoglobin of 13 gm. per cent, a leukocyte count of 1500 (normal differential) and a sedimentation rate of 90 mm. in one hour. Agglutination tests for brucellosis, psittacosis, toxoplasmosis, tularemia and typhoid and paratyphoid fever were all normal. The serum Wassermann had converted to positive at that hospital two years previously, after many negative Wassermanns in previous years. It was again positive on this admission to the St. Paul hospital. Blood cultures and also tests for lupus erythematosis were negative as were coccidioidin, histoplasmin and Mantoux (1:100) skin tests. For the first time at that hospital serial chest x-rays revealed a large nodular lesion in the right upper lobe, which continued to enlarge during his hospitalization. Routine x-rays of the chest taken during previous admissions there for various surgical procedures were normal, including the most recent one taken just a few months previously.

Because of the positive Wassermann, he was given 15,000,000 units of penicillin, without any change in his condition. A course of chloromycetin was given for possible typhoid fever, without benefit. Chloroquin was given for a suspected malaria, although blood films for malaria organisms were negative. His condition continued to deteriorate. Biopsy of an inguinal node revealed only fibrosis. A bone marrow biopsy showed hyperplasia in the smear and granulomata in the sections. Staining of the granulomata by the Ziehl-Neelsen method revealed no acid-fast organisms. A consultant in internal medicine thought that lupus erythematosis should be considered and steroid therapy was subsequently recommended and given. The temperature returned briefly to normal but then rose again and the patient developed ankle edema. Because of the bone marrow granulomata Isoniazid, Streptomycin and PAS were added to the steroid therapy. The temperature again returned to normal and remained there but the sweats continued. He gained twelve pounds and was discharged from the hospital. He returned to work for a brief period of time but the severe sweats continued and within two weeks he was again running afternoon temperatures of 102 to 105 degrees. Three weeks after discharge he returned to the hospital, where it was noted that his hemoglobin was now 5 grams per cent and the leukocyte count was 900 cells per cubic millimeter. He was given four blood transfusions in the course of the next few days. He experienced chills almost daily and described approximately five episodes of severe rigor which shook the bed and which lasted from one-half to two hours each. Shortly before transfer to the VA Hospital he experienced an episode of squeezing tightness of the chest which was accompanied by dyspnea and which was relieved by oxygen.

The past history revealed that he had undergone gastric surgery in 1923, a tonsillectomy in 1934, removal of a kidney stone through a left lumbar approach and also a left inguinal herniorrhaphy in 1953, and a right inguinal herniorrhaphy a few months before admission. He had suffered an attack of "the flu" one year before the onset of his present symptoms. The family history was non-contributory and the system review was negative except for an episode of jaundice several years previously. He had received several prostatic massages at some time in the past.

Physical examination on admission to the VA Hospital disclosed a well-developed, sallow man with evidence of recent weight loss. He spoke in a tired, monotonous voice and appeared to be in considerable distress. He was 6 feet, 2 inches tall and weighed 173 pounds. His temperature was 102 degrees F., his pulse rate 84 and his blood pressure 108/58. The sclerae were icteric. Blood oozed almost continuously from both nostrils. The tongue was coated and the breath was fetid. He was edentulous and a copious post-nasal drip was present. An ulcer was present in the left tonsillar fossa, on the right side of the lower lip and on the chin. "Fever blisters" were also present on the lips. Hard, slightly tender, enlarged inguinal and cervical lymph nodes were palpated. Post-tussive râles were heard posteriorly over both lung bases. The vital capacity was 3.6 litres, rapidly expelled. Well-healed scars of previous surgical incisions were noted over the abdomen, the groins and the left flank. The liver edge was palpable 14 cm. below

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the right costal margin in the mid-clavicular line and it was firm and tender. The edge of the spleen was palpated 6 cm. below the left costal margin in the mid-clavicular line and it was firm and non-tender. A pronounced, pulsatile palmar erythema was seen. The left lobe of the prostate was firm, non-tender and nodular. Hemorrhoids and varicose veins of the legs were present and the ankles were slightly edematous. The Babinski was thought to be positive on the right by one observer. The patient was very weak and walked with a staggering gait. The examination was otherwise within normal limits.

Laboratory tests revealed that the hemoglobin ranged from 10.9 to 9.6 gms. per cent on five determinations, with red cell indices on the latter determination as follows: mean cell diameter 7.4 microns, mean cell volume 95 cubic microns, mean corpuscular hemoglobin 28.5 micro-micrograms and mean corpuscular hemoglobin concentration 30 per cent. The hematocrit was 32 per cent and the red cell count was 3,350,000 per cubic millimeter. The leukocyte count ranged from 1350 to 2350 on four determinations, with normal differential counts. Two platelet counts were 14,000 and 40,000 per cubic millimeter. Two reticulocyte counts were 4.6 and 6.5 per cent. Direct and indirect Coombs' tests were negative. The erythrocyte sedimentation rate was successively 75, 83 and 80 millimeters in one hour, Westergren method. The bleeding time was seven minutes and the coagulation time was twenty-one minutes. A blood film for malaria organisms and two clot tests for lupus erythematosis were negative.

Two urinalyses revealed specific gravities of 1.017 and 1.023, albumin 2 plus and 4 plus and the tests for reducing agents were negative. The more concentrated specimen tested negative for bile, but contained 5 to 10 leukocytes per high powered field, 3 to 5 red blood cells per high powered field and 20 to 30 coarse granular casts per high powered field.

Liver function studies revealed that the serum bilirubin was 1.0 mgm. per cent on the one minute direct test. The total serum bilirubin was 2.6 mgm. per cent. The twenty-four-hour urinary urobilinogen excretion was 8.15 mgm. The cephalin flocculation was 4 plus in twenty-four and forty-eight hours. The thymol turbidity was 8.5 units. The zinc sulfate turbidity was 13.8 units. The prothrombin activity was 47 per cent. The total serum cholesterol was 105 mgm. per cent. The bromsulphalein retention was 27.1 per cent in forty-five minutes, when the serum bilirubin was as reported above and when the oral temperature was 101.6 degrees F. The total serum proteins were 5.9 grams per cent. These were subjected to electrophoretic separation with results as follows: albumin 2.5 grams per cent or 42.30 per cent, alpha$_1$ globulin .2 grams per cent or 3.55 per cent, alpha$_2$ globulin .5 grams per cent or 8.30 per cent, beta globulin .5 grams per cent or 8.30 per cent and gamma globulin 2.2 grams per cent or 37.55 per cent.

Evaluation of blood chemistries revealed that the blood urea nitrogen was 24.5 mgm. per cent on admission. It rose in seven days to 65.0 mgm. per cent on the day before death. The plasma CO_2 combining power on the same days was 30.2 and 7.5 meq./1. On admission the serum chlorides were 92.7 meq./1., the serum sodium was 130.4 meq./1. and the serum potassium was 4.5 meq./1. The serum alkaline phosphatase was 10.0 KAU and the acid phosphatase was 2.8 KAU. The admission fasting blood sugar was 106 mgm. per cent (99-100 mgm. per cent is the upper limit of normal in our laboratory.)

In a search for an infectious disease, the bronchial washings were smeared and cultured for acid-fast bacilli, but none were found. Blood cultured the day after admission grew out a non-hemolytic streptococcus in broth only. Another blood culture the next day was negative. Culture of a mid-stream urine sample grew out 3 000 colonies per milliliter of hemolytic and non-hemolytic staphylococci which were coagulase negative. Three purged stools were negative for parasites or their ova. Two routine stools were negative for blood. Cold agglutinins and serum agglutinins for brucella antigens and for typhoid H antigen were not present. Serum agglutinins for typhoid O and paratyphoid A & B antigens were all present in a dilution of 1:20. Heterophile agglutinins were present in a dilution of 1:14. A test for serum C-reactive protein was 3 plus. The VDRL slide test was positive in a dilution of 1:32 and the Wassermann complement fixation was positive. A test for serum cryoglobulins was negative. The treponemal immobilization test was positive.

An electrocardiogram was normal. X-rays of the chest, including planigraphy and bronchography, revealed a large homogeneous, lobulated mass in the right upper lung field in good focus from 9 to 14 cm. from the posterior thoracic wall. A mass in the right hilum was also noted. All the branches of the right bronchial tree could be filled with dye. One branch in the region of the mass was extremely irregular and narrow. Some cylindrical dilatation of the bronchi to the right upper lobe was present. The chest films of the patient's wife, son and daughter were reviewed and found to be normal.

The patient's hospital course was marked by fever with daily fluctuations between 99 and 102 degrees (oral). He was given prednisone daily during his evaluation in order to avoid sudden termination of the steroid therapy he had received elsewhere before admission. While being continued on steroid therapy, blastomycin, coccidioidin, histoplasmin and first strength PPD intradermal tests were administered however, and were all negative. Bronchoscopy was normal except for the presence of a non-ulcerated, smooth 5 mm. papule in the mucosa of the left main bronchus, the middle lobe orifice and the bronchus of the right medial basal segment. Microscopic examination of the washings revealed cells that led the pathologist to suspect a possible malignancy.

The tissue slide from the inguinal node biopsy performed at another hospital was sent for and reviewed and was interpreted as normal except for moderate fibrosis. Accordingly, on the fifth hospital day a biopsy of a left posterior cervical node was performed. By this time the patient's condition was deteriorating rapidly. His jaundice and edema had increased and he was troubled with anorexia, abdominal cramps, vomiting and diarrhea. He continued to perspire profusely, became dyspneic and apprehensive and on the eighth hospital day was obviously confused. His nose continued to ooze with a trickle of thin blood. His abdomen had become distended and very hard. Oxygen was administered but he complained of a smothering sensation with the mask on. On the ninth hospital day he was clammy and pale. His respirations became rapid and shallow and his pulse weaker and he finally expired before the cervical node biopsy report was received.

Discussion

Dr. Paul S. Hagen: I shall assume that everyone present has read this protocol. Approximately one month after this man's illness began, it was noted that he had hepatosplenomegaly and leukopenia. In another hospital a biopsy of an inguinal node showed fibrosis. There is no indication that a touch preparation of the node was examined before routine sections were made. Apparently the node was not cultured. A bone marrow biopsy showed hyperplasia and numerous granulomata. A Ziehl-Neelsen stain showed no acid-fast organisms. There is nothing in the protocol to indicate that the marrow was cultured on specific media for various organisms.

Dr. W. H. Hall: I don't believe the marrow was cultured.

Dr. Hagen: A chest film showed a nodular lesion in the right upper lobe. Before this conference began, I was told that a comparison of our own x-rays with those of the referring St. Paul hospital revealed an increase in the size of the area of the infiltrate. He was tried on various courses of antibiotics and yet had severe shaking chills. In spite of the antibiotic therapy, there was a progression of the pulmonary lesion.

A series of laboratory studies are described. From a hematologist's viewpoint, the essential points are that he had an anemia and a leukopenia. This was not a neutropenia, but a leukopenia. The reticulocyte counts were elevated and the platelet counts were low. The bleeding time was increased, compatible with the low platelet count. The anemia was normochromic and normocytic. I would like to defer further discussion of the laboratory test results until I have had an opportunity to see the patient's x-rays.

Roentgenology Resident: All the available films were taken within a few days of one another. Chest films disclose a rather round lesion which is fairly well demarcated from the surrounding lung (Fig. 1a). Some radiolucency within the lesion suggests cavitation. On the lateral projection the mass overlies the hilar shadows and for that reason we cannot make out its limits well (Fig. 1b). In addition, on the PA film we see hilar enlargement. Some scoliosis tends to accentuate the hilum. On planigraphy of the right upper lung field, one can see the minor fissure limiting the lesion inferiorly, so we know that this lesion is in the upper lobe. There is a lobulated appearance to the enlarged hilum, suggesting a hilar adenopathy (Fig. 1c). It does not appear to be present on the opposite side. The major branches of the bronchial tree are seen on several cuts and they appear patent and in their normal relationships. On bronchography there is fairly good filling of the bronchial tree. The lower and middle lobe bronchi appear normal. The upper lobe bronchi are well seen on the lateral film and show some changes suggestive of chronic bronchitis. There is slight irregularity to the bronchial wall. There are small filling defects suggestive of excessive secretion in the bronchial tree and the bronchi do not taper in the normal fashion. We can identify no bronchial connection between this lesion and the bronchi demonstrated. However, the mass lies adjacent to the pleura. It is not infrequent for one to fail to demonstrate a connection with a lesion in that position.

Dr. Hagen: Could you identify calcium within the subpleural or hilar masses?

Radiologist: No.

Dr. Hagen: Obviously we need further help from the laboratory in order to establish a diagnosis here. I want to point out once again that we are faced with a chronic fever unresponsive to antibiotics, including antituberculous agents. One needs to know whether antituberculous drugs were given for an adequate length of time to conclude that there was no response. Were they given for at least two months?

Dr. Nesbitt: I am sure they were not.

Dr. Hagen: Were they given for more than two weeks?

Dr. Nesbitt: That was about the total length of his stay in that hospital.

Dr. Hagen: The serum protein values may be of some help. On electrophoresis the albumin was moderately decreased and the gamma globulin markedly increased. In tuberculosis an increase in alpha globulin is more likely. The BUN was elevated. It is very interesting that certain agglutinations either were not obtained or were purposely not described. With this type of clinical situation there were certainly some other agglutinations that I think the physicians on the ward must have obtained.

A hemolytic anemia was possible because of the elevated reticulocyte counts. The thymol and zinc sulfate turbidities were elevated. These suggest that an immunochemical reaction could be responsible for the anemia and leukopenia. This is a possible explanation of the peripheral pancytopenia, because the bone marrow done at the other hospital was found to be hyperplastic. So the anemia and leukopenia were not due to depression of bone marrow activity but were more apt to be due to a hyperimmune mechanism. The protocol states that one blood culture was negative and one grew a non-hemolytic streptococcus in broth only. I wonder if we know how long these blood cultures were held.

Dr. Hall: They were cultured for 5 days.

Dr. Hagen: The blood cultures may have been thrown away too soon for other than routine pathogens. I don't see any mention here of the inoculation of laboratory animals and perhaps it is not fair to ask, because this may not have been done or the results may not have been reported by the time the patient died. I presume the urine was also cultured for just a short time.

Dr. Hall: Forty-eight hours.

Dr. Hagen: We may have missed an organism that is not one of the usual pathogens by not suspecting its presence before death and therefore not alerting the laboratory to the need for special culture techniques. The negative skin tests do not mean much in the presence of long term steroid therapy or in a patient that is overwhelmed by one of the organisms one is testing for. These tests are based on hypersensitivity, which may be lacking in such a patient. The patient might have been in an anergic phase of reaction to invasion.

How can we put this together? A possible explanation is one I am sure I am supposed to trip over, namely malignant lymphoma. One could postulate that he developed a lymphoma two years before he came in with a high fever and that the lymphoma produced an immune reaction which turned his serologic test for syphilis positive. Not only could the serologic test for syphilis change two years before a patient presented with fever due to lymphoma but also a positive Coombs' test could have been present the same length of time. We have recently had a patient that we had carried as an idiopathic acquired hemolytic anemia for almost two years. This was subsequently found to be due to Hodgkin's disease. Hepatosplenomegaly, leukopenia, probable hemolytic anemia and a hyperimmune process are perfectly compatible with a lymphoma. The serum protein electrophoretic curve with an elevated gamma globulin is also compatible with a lymphoma, although the ones we have seen have not had a marked elevation. The marked elevation of the gamma globulin was more consistent with an infection than with a lymphoma. There is a physical finding here that I think is of some importance. The liver was not only enlarged but was also tender, whereas the spleen was not tender. I have been trying to remember any patient that I have seen with a lymphoma who had a definitely tender liver. I wonder how tender this liver was. I think it is unusual to have a tender liver because of a lymphoma and I intend to take that as another sign that I had better get away from that diagnosis. Furthermore whenever one has a patient whose course is continuously febrile and whose fever does not relent with any antibiotic, a mycosis, with or without a lymphoma should be considered. I think that we have to give that diagnosis serious consideration in this patient. We should remember that it is not uncommon for patients with a lymphoma to develop an infectious disease and it is quite possible that this was the sequence of events with this patient. However it does not seem necessary to include the diagnosis of lymphoma when the entire picture can be explained by a certain infectious disease alone.

Disseminated tuberculosis is a possibility and is difficult to rule out. The failure of the patient to respond to antituberculous chemotherapy does not exclude this disease because he did not receive this treatment long enough for an adequate trial of therapy. He did indeed improve enough to be discharged from the referring hospital but his symptoms never left him completely and he had to return very shortly thereafter for his final admission there, because of a severe exacerbation of his illness. The negative skin test does not exclude tuberculosis for reasons already discussed. Acid fast organisms could not be found in his bone marrow granulomata or in his bronchial washings. While one would not expect to find the bacilli in his bronchial washings after miliary dissemination from a primary focus, one would expect to find them in this case because his pulmonary lesion is a large mass which is probably cavitary. A bronchial communication with the lesion would have to be present however. For these reasons, disseminated tuberculosis would not be my first choice.

A bronchogenic carcinoma with widespread metastases is a possibility but an unlikely one. The roentgenologist suspected cavitation of the pulmonary mass which, it is true, could be central degeneration of a tumor. However an infectious etiology is more likely. Suspicious cells were noted in the bronchial washings, but, again, these could just as likely be a proliferative response to infection. No occlusion of a bronchus can be seen on the planigrams and no "rat-tail" deformity characteristic of malignancy is seen on the bronchograms. If calcium were present in the subpleural or hilar masses, I would feel more secure in saying this was an infectious granulomatous process rather than a malignancy. On the other hand, granulomata were seen in the bone marrow and presumably no tumor cells were seen there. If the pulmonary lesions are also granulomatous they may not have been present long enough for calcium to form.

The ulcer in the tonsillar fossa in combination with the pulmonary lesion, hepatosplenomegaly, leukopenia, anemia, chronic unremitting fever in spite of antibiotics, night sweats, emaciation, and death within several weeks after the onset of symptoms are all highly suggestive of disseminated histoplasmosis. This man's residence in the Mississippi Valley is also consistent with that diagnosis and disseminated histoplasmosis is the diagnosis I am going to hang my hat on in this conference. The way to prove the diagnosis is to observe or grow the organism. This would not be likely with routine culture techniques and that is the reason I have been so inquisitive about the techniques used. One serologic test that is not mentioned in the protocol is the complement fixation test for histoplasmosis. For this test, we send our specimens to the Bacteriology Department of the Army Medical Service Graduate School at Walter Reed Army Medical Center in Washington, D. C. If this was done, there would not have been enough time for the result to have been returned to Minnesota before the patient expired. Therefore, since it would not have been available to the ward physicians before death, such a test may not have been included in the protocol.

Dr. Nesbitt: We felt that we were very fair with Dr. Hagen. We gave him all the information we had up to the time of death. Shortly thereafter additional facts became available. The first of these was the report on the cervical node biopsy.

May we have the diagnoses offered by the medical students?

Dr. E. B. Flink: The following diagnoses were suggested: coccidiomycosis, tuberculosis, sarcoidosis, amoebiasis, histoplasmosis, paroxysmal hemoglobinuria and Hodgkin's disease.

Dr. Hagen's Diagnosis

Disseminated histoplasmosis.

Pathologic Findings

Dr. D. F. Gleason: I think we have been treated to a fascinating demonstration of CPCmanship in this very beautiful case discussion. The permanent sections of the lymph node biopsy became available the night before the patient died. Histoplasma capsulatum was identified on the slides. Permission for an autopsy was initially refused by the patient's wife. However when the lymph node picture indicated that this case was an example of disseminated histoplasmosis, the wife was contacted again and was finally persuaded to grant an autopsy out at the mortuary on the day after the body was embalmed. Permission was limited to examination of the trunk only, however. The embalming process prevented us from obtaining accurate weights of the various organs and their appearances also were altered.

Upon gross examination an abscess was noted in the anterior segment of the right upper lobe. The abscess measured 4½ centimeters in diameter and had a liquid center, surrounded by a soft wall of connective tissue. Presumably this was the large round lesion seen on the x-rays. Numerous smaller soft nodular lesions were seen throughout the lung and another abscess, which measured one centimeter in diameter, was found on the other side of the chest in the left lower lobe. The liver was very large. Its weight was estimated at 4500 grams. The spleen was also very large, apparently weighing about 2000 grams. What appeared to be Malphighian corpuscles on first glance were actually small yellow abscesses or infiltrates. They were somewhat larger than Malphighian corpuscles, measuring about four millimeters in diameter. The kidneys also had very tiny yellow nodules scattered through them. The periaortic and hilar lymph nodes were enlarged and were quite soft and mushy. An incidental finding of chronic cholecystitis was also noted. The remainder of the organs within the trunk appeared grossly normal.

Microscopic examination of non-caseous material from the lung, stained with hemotoxylin and eosin, reveals myriads of macrophages, and even capillary endothelial cells, which contain intracellular organisms with a central body that stains with hemotoxylin and a clear capsule that does not take any stain. This Giemsa stain of the same section makes the capsule a little more visible. With this periodic acid-Schiff stain it can be seen that these intracellular organisms are Schiff positive. The central body stains a dark red and the capsule is also Schiff positive, staining a lighter shade of red. Similar intracellular Schiff positive organisms can be seen in the spleen (Fig. 1E), in the endothelial cells of the capillaries of the renal glomerulus (Fig. 1F), in these miliary nodules in the liver (Fig. 1G), in almost any lymph node (Fig. 1H) and in the bone marrow. The bone marrow is very extensively involved, even being necrotic in some areas. We wrote to the referring hospital for the bone marrow biopsy that reportedly contained granulomata, hoping to be able to identify the organism. We never received that marrow for review, however.

Dr. Hagen: The organisms you showed us in a lymph node were from a section, weren't they?

Dr. Gleason: Yes, these are all sections. We made some touch preparations from the lymph node and the organisms were seen by that method also.

Try to remember the relative size of the organisms you have just seen. We will now look at some sections from the center of that lung abscess. It is filled with necrotic debris. Under the high dry power and with routine hemotoxylin and eosin staining we find it difficult to identify a causative organism. Many similar

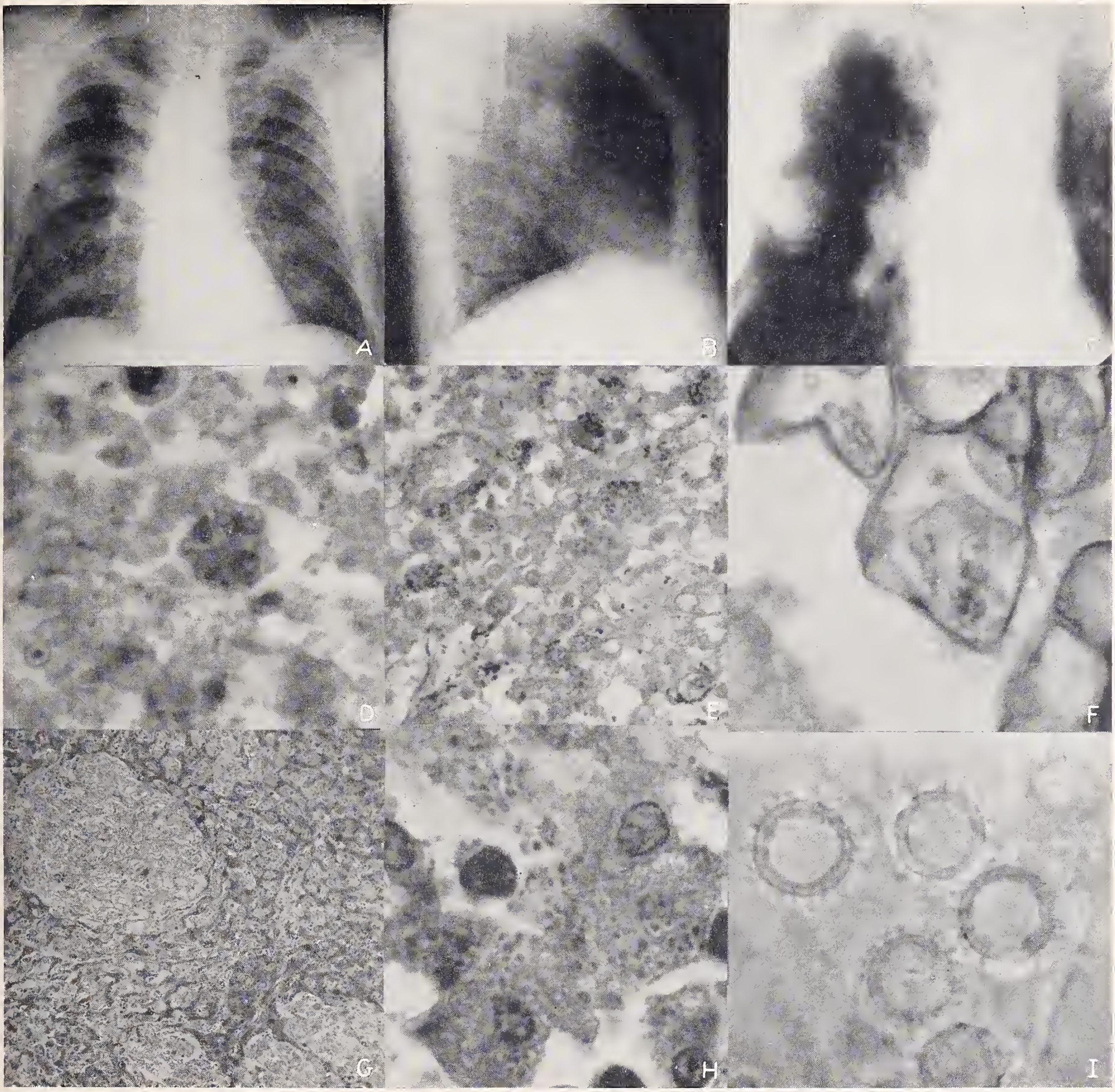

Fig. 1. Disseminated histoplasmosis. (A) Right subpleural and hilar masses on routine PA film of chest. (B) Lateral view with mass overlying hilar shadow. (C) Planigram showing lobulation of hilar mass and no visualization of calcium in either mass. (D) Puckett bodies in subpleural necrotic mass. (E) Intracellular H. capsulatum is seen in macrophages in the spleen and (F) in endothelial cells of a glomerular capillary. (G) Miliary nodules in the liver with numerous organisms seen. (H) Intracellular organisms in lymph node macrophages. (I) Tuberculate chlamydospores of H. capsulatum obtained on culture of a hilar node.

Photography by Medical Illustration Service, VA Hospital, Minneapolis. Selection of subject matter and supervision of photography by Martin A. Segal, M.D., formerly Assistant Pathologist, VA Hospital, and now Pathologist, Asbury Methodist Hospital, Minneapolis, Minnesota.

but smaller pulmonary nodules have been resected from other patients with similar findings on pathologic examination. In some instances they have been called "tuberculomas" or "idiopathic pulmonary granulomata, probably tuberculous." These "coin" lesions are usually rounded subpleural nodules with a fibrous capsule and a laminated necrotic center. Routine stains show no organisms and Ziehl-Neelsen stains show no tubercule bacilli. Puckett[1] has shown that many of these lesions, when stained with periodic acid-Schiff stain and examined under oil immersion, contain numerous encapsulated organisms which are now extracellular but which bear a close resemblance to Histoplasma capsulatum. The only objection to a diagnosis of histoplasmosis has been that the organisms will not grow in culture media.

Examination under oil immersion of the necrotic material from one of this patient's lung abscesses, which has been stained with periodic acid-Schiff stain, now reveals extracellular histoplasma organisms (Fig. 1D). However, many of them are almost twice the size of the intracellular organisms I have just shown you. Dr. Segal actually measured them and noted a statistical difference in the average sizes of the organisms in the center of these areas of necrosis. The central body in these large Puckett organisms is more hyperchromatic and the space between it and the capsular envelope is quite empty. The patient we are presenting in more detail today is the same one whose histologic findings only were described by Dr. Segal[2] before the Midwestern Section of the American Federation for Clinical Research at one of its meetings in Chicago. Using this patient, Dr. Segal was able to bridge the gap between typical intracellular forms of Histoplasma capsulatum and the bodies in necrotic debris which were described by Puckett. All variations of these forms, from the classic intracellular forms to those described by Puckett, can be identified in the sections from this one proven case. The Puckett bodies do not stain with hematoxylin and eosin in this abscess.

One of this patient's lymph nodes was cultured and it grew out organisms with a characteristic colonial morphology. These are "chlamydospores," which means spores with a mantle or protective envelope. The envelope is studded with periform tubercles (Fig. 1I). Tuberculate chlamydospores of this size and appearance are the proof positive of histoplasma in culture.

Dr. Nesbitt: Dr. Hagen is certainly to be congratulated for his very excellent discussion. Other things which he asked for and which did eventually become available after the death of the patient were the serological tests for fungi. The complement fixation test for coccidioides was negative. The complement fixation test for blastomyces was positive in a dilution of 1:32. The complement fixation test for histoplasma was also positive in a dilution of 1:32. The collodion agglutination test for histoplasmosis was positive in a dilution of 1:8.

The bronchoscopic washings also grew out Histoplasma capsulatum, the report being received some time after the death of the patient.

Dr. Johnsen: It is interesting to note that the first case of disseminated histoplasmosis ever described in the United States was that of a Minnesota woman. I believe this case was reported by Dr. C. J. Watson[3] of the University of Minnesota, who did the autopsy. The disease was first reported by Darling as a kala-azar type of syndrome in Panama. Dr. Watson[4] later described the pathology of the disease in detail.

Dr. Hall: The complement fixation test was done at Walter Reed Army Medical Center. We have frequently received reports of cross reactions. Complement fixation tests positive in titers of 1:8 or 1:16 for blastomyces in cases that we think otherwise are histoplasmosis by skin test and by clinical features have been frequent. This present patient is the only case that we have had that has been proven by culture to be histoplasmosis. Most of our other cases have been those with a solitary granuloma. These contain the large extracellular organisms and they have failed to grow in culture.

The positive TPI test is a surprise because this patient was questioned about syphilis several times and he was said to have denied any previous lesions or any treatment for syphilis. Many people regard the TPI test as being absolutely specific for syphilis. It is, however, a test which remains positive even though the disease becomes inactive or is treated successfully. I don't know how to explain the two-year history of a positive Wasserman and a positive TPI test.

Dr. Johnsen: We don't know how long this man had been host to the histoplasma organism. I wonder if it is not possible that he was harboring the organism two years ago and that the conversion of his VDRL, Wassermann and TPI tests was another example of a cross-reaction to histoplasmosis. Perhaps Histoplasma capsulatum contains antigens that are not only chemically similar to those of Coccidioides immitis and Blastomyces dermatitidis (which we know to be true because of skin tests and serologic tests) but are also chemically similar to those of Treponema pallidum.

Dr. Hall: I think there might have been some value in examining his blood film more closely. We might have seen the organisms in his peripheral blood. No organisms were found in the lymph node from the other hospital but I suppose they could have been there and could have been missed. The only tissue from which we got a positive culture was a hilar lymph node. I would also like to say that if a sizable piece of necrotic material can be obtained, even though it has been in formalin, it is worthwhile to wash it and to cut into the center of it to obtain material for a culture. This case proves that all of the organisms may not be killed by the formalin, because of incomplete saturation of the tissue.

Dr. R. E. Smith: What was the working diagnosis before death?

Dr. Nesbitt: The diagnoses considered by the clinicians on the ward were malignant lymphoma, bronchogenic carcinoma with metastases and a possible Budd-Chiari syndrome. Dr. Horace Zinneman held out for histoplasmosis.

Dr. Smith: I would like to hear Dr. Hall's comments on therapy.

Dr. Hall: There isn't any therapy.

Medical Resident: Does anyone feel that the organism became more disseminated because of steroid therapy?

Dr. Hall: Well, that is possible. Certainly, the organisms were very numerous, being found even in the endothelial cells of the capillaries. Not having effective chemotherapy, I think it is reasonable to think that the steroids might have caused further dissemination. However, one can see a similar course without steroids. He already had the symptoms and signs of dissemination by the time he was admitted to the referring hospital, which was before steroids were given there.

Dr. Zinneman: I would like to ask Dr. Gleason what the adrenal glands looked like.

Dr. Gleason: A few macrophages in the sinusoids of the adrenal cortex contained the organisms but there wasn't any necrosis or focus of inflammation.

Dr. Hall: Many cases of disseminated histoplasmosis die with Addison's Syndrome because of destruction of both adrenal cortices.

Dr. Hagen: The relatively low blood pressure and low serum sodium made me wonder about a secondary adrenal insufficiency here.

Dr. Nesbitt: Dr. Flink is interested in the impression of the medical student who really did a very fine job in working up this case. He included histoplasmosis in his differential diagnosis.

Final Anatomical Diagnosis

Disseminated histoplasmosis.

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Editorials

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TRENDS IN MEDICINE

It is trite but nonetheless true to say that we are living in a rapidly changing era. Medicine—both in health and in disease—has changed more rapidly in the past two decades than in all of its history, and the end is not in sight. So rapid and profound are these changes that those of us who are living through them, like passengers in an express train, have only a kaleidoscopic picture of our changing surroundings. It is my purpose in these comments to discuss the present status of medical care, to outline some of our current problems, and to enthuse about a new and idealistic program for the prevention of disease.

As far as physical illness is concerned, the advent of chemotherapy has virtually eliminated most infectious diseases and will probably soon reduce the few remaining to unimportance. Malignant illness is not immune to our attack and may soon succumb. Even degenerative illness shows promise of conquest. Surgical advances grow ever bolder and more imaginative. I believe we shall even see a completely synthetic heart—doubtless of plastic—replacing a damaged one, activated by a pocket battery with a rheostat to permit greater emotional or physical exertion! Scarcely an organ in the body has not been proudly removed and often replaced by a graft of some type, and all sorts of trauma yield to the skill of the surgeon—internist — anesthetist — physiologist — et al who comprise the modern therapeutic team. In the exciting drama of modern medicine, we bemoan the general practitioner and write diatribes against the age of specialization—saying much the same that was said forty years ago or longer. We have coined a new word "generalist," though what is encompassed in the practice of this modern superman is not defined. Intelligently we regard the family doctor, usually an internist, as a conductor of an orchestra. It is he who controls the medical destiny of his patient, but it is he who calls upon the skills of others when his own are inadequate. This would appear to be the acme of complete medical care, not only for the individual, but also for the family—for we know that no man is an island but an archipelago with parents, spouse, children, friends and associates, boss and subordinates influencing every symptom which he has.

The field of emotional illness has expanded also. Not only do we conquer or control intractable insanity with weirdly formulated chemicals for which we have coined jaw-breaking names, but we also feel ourselves to be pioneers in discussing "psychosomatic illness," which is in fact as old as man himself. I believe Paul White has described the mind and body as bearing the same relationship as an electric current and a magnetic field—neither can be altered without affecting the other. Scarcely an illness or an accident lacks its emotional response; scarcely an emotional storm or disappointment, irritation or ambition, fails to have some associated physical reaction, be it diarrhea, pain, headache, sweat or palpitation. Let us not, therefore, speak of mind and body as separate but as co-ordinated apparatuses. They are in reality inseparable as salt and water in the ocean.

Equally important, though less susceptible to observation and analysis, is the spirit. No physician worthy of the name but respects the power of courage to heal, and of fear to destroy. No physician has cared for the sick without bowing his head to the power of prayer and faith to overcome impossible obstacles. Any thinking person respects and acknowledges the healing and refreshing power of meditation, solitude, faith and prayer.

It may seem strange to bring faith, ambition, and specialization together with antibiotics, surgery, and the family doctor. Yet this is the essence of care of the whole man. We cannot, as practitioners of the healing art, content ourselves with prescribing pills or injections, with amputating or replacing, or with auditing the recollections of the conscious and the sub-conscious. It is properly our function to investigate and to understand family relationships, economic and social pressures, and the religious faith of those who entrust their health—in its broadest sense—to our care and to formulate from this, concrete and practicable recommendations.

First of three editorials on Trends in Medicine.

The periodic health examination when properly done is a tremendous advance in medicine. By this examination, the physician may anticipate trouble, and by anticipating may prevent. But the scope of such an examination is awesome. Not only must the physician take a complete history, not only must he do a thoughtful physical examination, but he must also gain by question and intuition some knowledge of the emotional life, the social career, the economic problems, the ambitions, *vis-a-vis* the accomplishments of his patient. Much of this knowledge comes by osmosis during the examination. In smaller communities, the doctor's social contact with the patient fills other gaps. Often the more pressing problems are volunteered, provided the physician is receptive and appreciative. But there is always a residue which must be deliberately sought if we are to do the best job. The money fears, the shame over past errors, the feelings of inadequacy, the problems of sexual relationships, are seldom offered unless questioned. Last but not least are the indispensable laboratory tests and x-rays. The latter raise the dilemma of irradiation hazard in the physician's mind.

Can such a complete health examination ever be actually accomplished? I suspect the answer is no, but completeness can be approached if we bear in mind the complexity of the problem, and if we are as sensitive to things unsaid as to those spoken. But we must always realize that the best preventive medicine should not end with cancer prevention, detection of early diabetes or hypertension, or with the recognition of existing illness in an early stage. The best in preventive medicine means a positive approach to good health, an approach which goes beyond illness, which suggests and implements things which can and should be done to attain maximum health, optimum pleasure and productivity. This approach must include a scientific medical examination, a delicate and perceptive emotional evaluation, and spiritual or intellectual feelers equally delicate and equally important. Only through this approach to the whole man can the physician discharge his treasured responsibilities. His final step, however, must be to offer positive recommendations rather than a statement of negative findings.

For this reason, the family physician holds the highest position in medicine which a man can have. But this is also why medical care is ever changing and ever challenging. This is why we must be ever alert to growth. This is why one speaks of "practice" of medicine.

Charles S. Houston, M.D.
Aspen, Colorado

GOVERNMENT

Government exists for the people. And the nature of the population has a major effect on the problems of government.

One of the most important factors in our rapidly changing society today is the growth in our population. In any planning for the immediate future we must take into consideration, not only the increased numbers, but the changing composition of this total population. Sensible decisions cannot be made unless the following facts are considered.

In 1946, Minnesota's total population was 2,734,000. In 1955, it was 3,174,000. In 1966, it is expected to be 3,600,000. If we keep government services only at the present level, it is obvious that the volume of services will have to be increased to keep pace with our increasing numbers. And if—as has been true in the past—the people want more and better services, such as more years of education for more of our children, the services will have to be increased even more.

Another important fact about our population is that it is changing in nature as well as in total numbers. The two age groups that show the greatest increase are those under twenty-one years of age and those over sixty-five.

One calculation of changes during the fifteen-year period between 1950 and 1965 shows that, while our over-all population would increase 22 per cent, those under twenty-one years would increase 38 per cent, and those over sixty-five years would increase 35 per cent. In this same period, the number of people in the most productive years between twenty-one and sixty-five would increase only 9 per cent. And we all know that it is the young and the old that make the greatest demand on government services. We must plan to meet that demand.

The increased proportion of children and youth makes it imperative that we expand our educational facilities and do everything possible to secure enough well-qualified teachers. We need to build more schools and classrooms.

Another trend that we all welcome is that an increasing proportion of our young people are

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Third in a series of editorials on Government.

seeking higher education. We hope and expect that this trend will continue, but it presents our colleges and university with the very real problem of financing rapid expansion. Expenditures for education are, in my opinion, the wisest investment we can make. But we must honestly face the problem of raising the money needed to make that investment.

The increasing proportion of older people likewise creates a financial problem, primarily because of the increasing cost of providing medical care for the recipients of Old Age Assistance.

In spite of the increase in the total number of citizens over sixty-five years of age, the number who receive Old Age Assistance is steadily declining. In 1946, 54,000 were receiving OAA; in 1956 this number was 51,500; and we estimate that about 45,000 will be receiving assistance in 1966. This decline is due to the progressively increasing number of people receiving old age and survivors' insurance benefits under the provisions of the Social Security Act. Thus our expenditures for maintenance under the OAA program do not create a serious problem.

But the continuing and increasing need of our older group of citizens for more medical care has made this program increasingly expensive since 1945, when unlimited medical care for Old Age Assistance recipients was authorized from state and county funds.

In 1946, only about 17 per cent of OAA recipients were medical cases. Today, 47 per cent receive medical care, and it is apparent this trend will continue. During the past ten years the cost of medical payments has risen from $1,500,000 in 1946 to $16,000,000 in 1956, and an expected $17,000,000 in 1957.

Over 70 per cent of these medical payments represent nursing home and hospital care costs. Today, about 4,500 nursing home beds are occupied by OAA recipients. About 1,000 old people who are now patients in mental hospitals could be cared for in nursing homes if they were available. We estimate that in 1966, 9,000 nursing home beds will be needed for OAA recipients alone.

We are gratified at the increased life span of our people. We are determined that all of our older citizens shall get the medical care they need. But we must honestly face the problem of increasing costs, and find some way to meet them.

Orville Freeman
Governor of Minnesota

EMPHASIS ON DIETARY SOURCES OF THIAMINE

Concern for the trend toward low levels of thiamine in the American diet is being expressed with increased frequency. USDA nutritionists admit that the amounts of thiamine recommended by the Food and Nutrition Board of the National Research Council are difficult to achieve without careful planning.

Dr. Faith Clark took note of this when she addressed the Nutrition Education Conference in Washington in April, 1957. She said:

> ". . . Thiamine levels are little related to family income. Sources of thiamine in diets are chiefly grain products, meat (especially lean pork) and milk. Although consumption of milk and meat increases with income, that of grain products and of lean pork cuts generally decreases.
>
> ". . . Various nutrients are affected differently by changes in the economic situation. For example, a nutrition education program for a period of prosperity would not need to emphasize protein or niacin. When people in this country have more money to spend for food, they are likely to buy meat. *Thiamine,* on the other hand, *would need to be emphasized in a nutrition program for a prospering economy.* The high income diet is likely to mean a shift away from grain products and pork and the foods chosen to replace them are not equally good sources of thiamine."

This change in dietary pattern is not entirely due to our present prosperity. Dr. Herbert Pollack, in addressing the New York State Dietetic Association meeting in April, 1957, reported an increase in the incidence of thiamine deficiency and beri-beri among diabetics who had been restricted in their use of bread. This development, as a result of dietary restriction, strikes a sharp note of caution to members of the medical profession: When making dietary recommendations, due consideration should be given to the inclusion of substitute sources of thiamine.

Says Dr. Russell M. Wilder, world famous authority on vitamin deficiency disease:

> "I worry most about deficiency of thiamine, because some time ago I was engaged in a very extensive study of the clinical effects of diets low in thiamine and have experimental proof that deficiency of this vitamin, a degree of deficiency so mild as to defy detection by the doctor, may provoke disturbances that are tremendously significant. Courage and emotional stability, with other qualities of character, deteriorate in thiamine deficiency before the nerves to the arms and legs are in any way affected; that is before neuritis has developed on which the doctor depends for a diagnosis."

The present emphasis on obesity has also caused many people to eliminate bread from the diet or restrict its use because of the erroneous concept that bread is a fattening food. This trend contributes to the dietary changes reported by Dr. Clark. Actually, bread contributes a higher proportion of one's needs for protein, thiamine, niacin, calcium, and iron than it does of one's need for calories. Because of this, bread serves a useful purpose in the reducing diet.

As a means of assuring a more adequate distribution of thiamine, riboflavin, niacin, and iron, the enrichment program was started in 1941. Dietary surveys had indicated that many diets did not provide adequate quantities of these nutrients.

The enrichment program has successfully accomplished its purpose. During the war years and for several years thereafter, symptoms due to a deficiency of the enrichment nutrients practically disappeared. In Newfoundland, an enrichment program contributed to the eradication of beriberi, to a 50 per cent decrease in infant mortality, decreases in the crude death rate and in tuberculosis. Nutritional deficiency states, once prevalent among the inhabitants of Chicago's Skid Row, have virtually disappeared. Kark et al., attributed this improved nutritional status to the enrichment program when a study of the food habits of the Skid Row population revealed one variable in their diet: enriched bread.

Avitaminoses, once prevalent among groups recognized as having poor diets, are now appearing in those who formerly had good eating habits. This finding reflects the shift, in our present-day economy, from food sources of the B vitamins, particularly thiamine. For our period of prosperity, Faith Clark has pointed out the need for emphasis on thiamine in nutrition education programs. The key to the role of enriched bread in such programs is revealed by a glance at its nutrient contributions: Six slices of enriched bread supply 23 per cent of the thiamine, 12 per cent of the calories, 18 per cent of the protein, 16 per cent of the calcium, 30 per cent of the iron, 16 per cent of the riboflavin, and 21 per cent of the niacin recommended by the Food and Nutrition Board as daily allowances for the twenty-five-year-old man.

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American Institute of Bakinc

CAN YOU BE INSURANCE POOR?

Plans for Retirement

In planning for retirement or a "slowing down," the physician cannot include Social Security or a pension provided by an employer as his neighbor can. Therefore, the degree of his retirement depends upon his own planning during his active years.

The physician may feel it unnecessary to plan for retirement, calling to mind examples of elderly men, enjoying their continued activity and maintaining their reputations as competent practitioners years beyond the normal retirement age. It is an advantage of a profession, of course, that an individual can select his own retirement age—or can he? Unfortunately, compulsory retirement is not limited to employes of large firms, but in applying the term to the physician I am referring to another type of compulsory retirement, that brought on by failing health. Often such failing health occurs after disability income insurance has terminated. In discussing this problem recently, one physician said, "I don't know whether I shall want to retire, but if forced to retire, I want to be in a position to do so and still maintain my own independence and self respect." Doesn't that seem to be a sensible approach to the problem? That seems to be the feeling of many professional people.

It may seem discouraging to plan for retirement without the aid of Social Security or a pension with progressive income taxes making it more difficult to save and with increasing life expectancy following retirement. However, many physicians are not aware of some important tax factors written into the Internal Revenue Code of 1954. These tax factors eliminate entirely, or greatly reduce taxes on income at retirement. The three main factors are:

1. A new method for determining the taxable portion of annuity income.
2. A special retirement income credit for persons not covered by Social Security, and
3. Double personal exemptions for individuals age 65 or older. An example will illustrate how these tax credits offer real encouragement to planning a good retirement program.

In a previous article, an example was cited of a young doctor who set up a life insurance estate of $100,000 by purchasing $25,000 permanent life insurance and using a $75,000 term insurance rider. The $75,000 term rider is convertible regardless of health, in this case, within the ten years. This doctor probably will decide to make the $75,000 term or a good portion of it a permanent part of his estate by converting it when income permits. He may decide also to start planning a systematic retirement program. Using this policy as a stepping stone, he may build up as much assured life income as $12,000 per year at age sixty-five, without Federal Income Tax, if he and his wife are both sixty-five, because of tax credits afforded under the present tax code. If he is single, divorced, or a widower at sixty-five, he may have as much as $9,000 per year free of Federal Income Tax. The exact amount of life insurance or annuity income that one may have free of Federal Income Tax will depend on several factors.

As the physician approaches retirement and prepares to hand over his privileges and responsibilities in his chosen profession to his younger colleagues, he probably will list his financial objectives in this manner: (1) Adequate retirement income for as long as it will be needed, (2) adequate income for his widow if he should be the first to die and (3) the transfer of the unexpended estate to his children and grandchildren. In line with these objectives he probably will want his retirement income guaranteed for a minimum period of years for the benefit of his widow if he should die before the end of that period, but income continuing during his lifetime after the minimum period. An example of this is the income for 120 months certain but for life thereafter.

It may be advisable to have paid up life insurance adequate to cover estate clearance costs upon his death including administration costs and death taxes, and adequate to provide his widow with sufficient income should she survive him. With adequate lifetime income assured for himself and his wife, the physician may feel that he is in a position to transfer portions of his estate to his children while he is still living and be able to enjoy seeing the children and grandchildren benefit thereby. A further incentive to making such gifts may be found in the difference between the gift taxes and death taxes.

Certainly, it will be advisable for the physician to consult with his own life underwriter as well as his attorney and possibly the accountant and trust officer in mapping out the estate plan best suited for his particular situation. There is no one medicine or wonder drug that will cure all ills and there is no one estate plan that will solve the problem for everyone.

Whether it be the young physician beginning practice, the established practitioner or the doctor approaching retirement, who, would you say, is more likely to be "insurance or property poor"? The man who establishes financial goals and objectives early and is willing to earmark a reasonable portion of his earnings to achieve these goals by proper planning? Or the man who has not mapped out his financial plans in advance but is earmarking the same or a greater amount of his earnings for the general purpose of building an estate? The latter man likely will forget, too, that patients and financial programs both need regular check-ups.

The phrase "insurance poor" throughout has been used in its literal context. But, when someone says that Dr. A, in providing for his family's security, has become "insurance poor," would it not be appropriate more often to use the term "insurance rich?" He can say he is a wealthy man because he has, on one hand, peace of mind and, on the other hand, an unworried attitude toward the financial pressures of today. His plan has enhanced his family's standard of living instead of competing with it. If there were no such thing as insurance, he could not in conscience spend as freely as he does for fear of jeopardizing his own and his family's future.

Herbert F. Mischke, C.L.U.

CONFESSIONS OF A HOBBYIST

Conclusion

The varied activities of which I have been writing have brought me infinite pleasure, and very little money. An occasional fee for a lecture on music, or an illustrated lecture on wild flowers

—an occasional sale of a pair of the little tables I've made in the shop, though most of these have been given as wedding presents to graduates of the Saint Paul Academy—a few dollars for written articles on radio—that is all. I instinctively shy away from definite orders to make things, partly because I don't like business transactions with friends, and partly because I don't want to sell anything that isn't perfect. But some of the activities have saved a lot of money around the house. For instance, the turning of an old barn into a summer place for my older son and his family made use of every bit of carpentry experience I had ever had, and some that I'd never had. There wasn't a right angle or a plumb line in the whole building, which made it a nightmare. Now it is a habitable house, with three bedrooms and a bathroom where the hayloft used to be, a modern kitchen in the old harness-room, and a pleasant living-room where the stalls used to be. Happily, I had another man helping me on this job—an old-timer, who knew more than I did about things like shingling, or fitting window-sash.

Certainly, what might be called "controlled versatility" enriches one's life. It also brings the painful realization that two things are rigidly rationed—time, and physical and mental energy. I quote from an address my father once made to the Yale Freshmen. " 'All things,' says the Emperor Marcus Aurelius, 'are implicated one with another, and the bond is holy: and there is hardly any thing unconnected with any other thing.' Thus there is scarcely a fragment that may not be fitted into the mosaic of life. . . ." Both the Emperor and Dean Briggs knew what they were talking about.

Life is full of curious surprises. The thing I believe I have done best and enjoyed most is the teaching of mathematics to adolescent boys, my unorthodox preparation having been a college course consisting mostly of classics and music! But into the mosaic of teaching, and of schoolmastering in general, almost every apparently unrelated hobby has come in somewhere. The reader may suspect, by this time, that I have no regrets for the hobbies. Now I must stop writing—there is a color print that doesn't satisfy me, and I must go to the darkroom and make it over!

John deQ. Briggs

AMERICAN HEART ASSOCIATION RESEARCH

The American Heart Association has announced it has channeled a total of $29,000,000 into research studies of cardiovascular diseases during its first ten years as a national voluntary health agency. More than $1.1 million of the total has been spent in Minnesota alone, according to the Minnesota Heart Association, one of fifty-six affiliates.

Marking its first decade of assault on diseases of the heart and circulation, American Heart Association is spending $7,000,000 on research during the current year alone. Twenty-five per cent of all funds raised by its affiliates are sent to national headquarters, which reallocates grants to scientists throughout the nation.

In Minnesota alone this year, fifty researchers at seven institutions are sharing $317,762 allocated from the 1957 Heart Fund drive. Sixty per cent of each Heart Fund dollar is spent on research, announced Dr. Milton Hurwitz, president of the Minnesota affiliate.

The American Heart Association plans a "massive assault" in the next decade on hardening of the arteries, which is responsible for more deaths and disability than any other ailment.

The 1958 Heart Fund drive is being conducted throughout the nation February 1-28. The goal in Minnesota is $505,000.

E. H.

ADDENDUM TO TUBERCULOSIS CASE FINDING PROGRAM FOR MINNESOTA

Your Tuberculosis Committee recently presented a seven-point program for tuberculosis case finding dealing primarily with tuberculin testing as the major substance of the program (see Minnesota Medicine, October 1957, page 732). It is feared by some that though the tuberculin test is definitely important, over-emphasis on this one procedure may encourage neglect of certain basic principles of case finding and control.

There is a saying, "Tuberculosis is where you find it." It would appear that many if not the majority of cases of active tuberculosis coming under care even today are first suspected by the private physician on the basis of some point other than the tuberculin test. Certainly any experienced physician will recall patients in his own practice discovered through application of tuberculin. He will also recall cases in which suspicion was first aroused through a particular complaint heard in the office, home or hospital; or through some physical sign; or through an x-ray taken either for specific or general screening purposes; or through unexpected laboratory findings. Neither any one of these nor any other diagnostic hint of tuberculosis can safely be ignored by the conscientious physician or health advisor. On the other hand, such a point of suspicion does not by itself constitute a diagnosis nor even complete case finding. The isolated tuberculin reaction, questionable x-ray, or other finding suspended in mid-air and unsupported by other evidence is of itself rather fruitless. All too often in past surveys a person has been found with positive Mantoux reaction, x-ray finding or other significant sign of disease only to be hung up in a statistical exhibit with nothing further done, but any such point of suspicion which triggers a full case study may be health-saving or life-saving.

What constitutes such a plan of study? In brief outline, the following is suggested:*

1. A thoughtful history with searching questions regarding tuberculosis contact, symptoms, previous diagnoses, former positive or negative tuberculin reactions, x-ray study, operations, etc.
2. Comparable physical search for tuberculous foci.
3. Meticulous application and interpretation of the Mantoux test, and perhaps later re-application.
4. Judicious x-ray filming, routine or special, of the chest or other body areas under suspicion.
5. Competent bacteriological study of body secretions, fluids, and tissue specimens by microscopic, and more particularly by cultural and animal inoculation methods.

Any one of the above procedures may constitute a trigger mechanism for case study but only the combination of procedures constitutes balanced case finding.

In all such study we must remember that no one test is completely infallible in demonstrating tuberculous disease. The finding of tubercle bacilli has in the past been considered positive proof, but today bacteriologists occasionally doubt their own findings. Over-reliance on the x-ray film misleads us fairly frequently and the tuberculin reaction can likewise be contradictory though far less often. Yet one sanatorium in the State has had several proven cases of active tuberculosis in recent years in which tuberculin reactions were negative during at least part of the period of activity.

This is not to minimize the real value of such test procedures and survey methods as we have. As we narrow down the incidence of tuberculosis today, these procedures need to be applied more widely and strategically than before. The ideal case finding survey still would consist of the following steps in order:

1. Mantoux testing of all those not previously known to be positive reactors.
2. Thorough periodic clinical review and chest x-ray filming of all positive reactors and known cases throughout their lifetime.
3. Bacteriologic, special x-ray and other special laboratory examinations as clinical study may indicate.
4. Faithful reporting to the State Department of Health of all known cases of tuberculosis.

Unfortunately, we cannot routinely or even frequently reach our public health aim directly; we are forced into detours. In the present instance a practical answer is to reach annually as many in the community as possible but in particular to aim at (1) groups with known high incidence of tuberculosis by virtue of racial, economic, social, occupational or other factors, or known contact to active cases (this should include special investigation of the record of any case first reported by death certificate; also thorough study of the contacts of such cases); (2) groups known to be of special epidemiologic concern to the rest of society, such as food handlers and those occupationally close to children and adolescents.

Our plea then should be for a balanced program, a broad and persistent one, attacking at all vulnerable points, not only at one or two. And finally, to remember that there is no substitute in tuberculosis case finding for the alert physician; for the man with a high index of suspicion, the man so thorough as to be dissatisfied with the performance of one isolated test unsupported by broader case study.

R. W. Backus, M.D.

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*For fuller outline, see Report of Council on Public Health reprinted in Minnesota Medicine, Feb., 1957, Page 137.

President's Letter

MEDICAL PRACTICE IN A CHANGING WORLD

Immersed in a busy practice, we doctors presume that the circumstances under which we practice are unchanging and, in fact, unchangeable. The private practice of medicine, whether alone or in a group, in this country has seemed the ideal way to care for the sick and to improve the health of those who are apparently well. In the United States, the freedom of the sick to choose his own physician has been a jealously defended right. Conversely, the freedom of the physician to choose his patients and his type of practice without dictation or interference by third parties has been a foundation on which American Medicine has been built.

These freedoms have been lost in several foreign countries and are being attacked in this country. Their value is unquestioned by those who remember the principles on which our government and our culture were founded, but there are many who now would change our way of living. The medical profession has been called conservative, not to mention reactionary, in its efforts to maintain freedom of choice, but the most liberal political idea ever proposed is the American idea of personal, individual freedom of choice in personal, individual matters. The care of one's personal health and the climate of medical practice are basic factors in the general freedom. Change in these basic factors is a major step toward change in our whole way of living.

As we carry on our practices and try to keep up with the demands of our patients and the advances in medicine, we feel that nothing more can be expected of us. Knowing our way of practice is the best, we expect it not to change; however, it can change and is changing. How radical the change may be is not determined only by ourselves or by our patients, but by the general pattern of political and economic life. It is determined by the County Commissioner and the County Welfare Boards and management-labor contracts. It is determined by the State Government, the State Welfare Boards, the State Legislatures, through the State Income Tax Commissioner, the State Board of Medical Examiners, and the medical practice acts. It is determined by the insurance companies, the Blue Shield, and perhaps by co-operative clinics and other "third parties" to medical practice. It is also determined by the Federal Government through Medicare, the Veterans Administration, Social Security, the Armed Services and even the Department of Agriculture through the Food and Drug Administration.

Our relations with all these forces are not individual. They can only be carried on through the profession as a whole; more specifically, only through our County, State and American Medical Associations. Our Medical Associations can do for us what we want only if we, individually, tell them what we want. That can be done only when we are there to discuss, to vote and to elect. The voice of the profession may be the president of the society, but the policy is made by the boards of directors of the County Societies and by the delegates to the State Association and by their delegates to the American Medical Association.

Election time is here. Choosing a director or a delegate is serious business. When you find a good one, keep him if you can, because he is the one who decides policy, who is the power in the association and is part of the group which deals with those who determine how you shall practice; talk to him, know him, know what he and the societies are doing, and then support him.

Horatio B Sweetser

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

MEDICARE PROGRAM IS ONE YEAR OLD

The military's medicare program was one year old on December 7. To date, according to the Defense Department, the government has paid more than 300,000 physicians' bills amounting to $22 million, and over 200,000 civilian hospital bills totaling $21 million. While the combined total is considerably under the estimated $76 million a year for the program, the Office of Dependent's Medical Care points out that there is a backlog of claims. In all likelihood, the President's budget going to Congress next month will ask for around $76 million.

Administrative costs, according to OCDM, have been running around 3% of total expenditures. Almost 40% of medicare patients have been maternity cases. The Air Force leads the services with 41% of eligible dependents participating; then the Navy, with 32%; Army, 25%, and Public Health Service, 2%.

The medicare program was enacted into law on June 7, 1956, and became effective six months later.

OCTOBER BUSY MONTH FOR FDA IN ENFORCING LAWS

The Food and Drug Administration reports that in October of 1957: 121 voluntary actions to comply with the law; seizure of 561 tons of contaminated foods in 96 Federal court actions; convictions in nine cases involving illegal over-counter sale of prescription drugs; one court conviction and granting of injunctions in two cases involving interstate shipment of insanitary foodstuffs and certain self-treatment devices. Included in latter category was a vibrator which was represented as a treatment for cancer and heart ills.

UNCLE SAM, M.D., PROSPERS AS HE PRACTICES

Nearly one-half of all medical costs are paid for with tax money, states Dr. Thomas H. Alphin, of the AMA's Washington office.

"The physicians in this country can no longer afford to scorn their civic duty and duck their responsibilities to be part of the Federal government," he declares.

Here's why. The amount proposed to be spent for medical care and research in 1957-58 is $3 billion, more than 10 per cent of the non-defense Federal budget and over 25 per cent of the total medical budget of the whole country, including all costs, physicians' fees, hospital bills, drugs, hot water bottles—the whole ball of wax.

And, says Dr. Alphin, when the state and local government spending is added to this amount it is almost double—nearly one-half of all medical costs are paid for with tax money: $6 billion a year total, half Federal, half state and local, and amounting to half the Nation's entire medical cost!

Biggest spenders for medical care: the Vets Administration, the Dept. of Defense, and the Dept. of HEW. "They digest dollars at the rate of $6½ million a day, $250 million per hour, nearly $75.00 every time a clock ticks!"

With the beginning of increased vendor payments to OASI benefits, Dr. Alphin states, the medical care expense of the Federal government will be increased by possibly as much as $150 million annually, this money to be matched by the states, for a resounding total of $300 million a year.

Tax dollars are not the only concern of harassed physicians. There are millions of people—patient—involved in the story of Uncle Sam, M.D.

Says Alphin: "Legislation currently proposed would provide for partial Federal payments for sickness and injury of Federal employes and their dependents, about seven million in addition, which would make a total of nearly 50,000,000 people entitled to free government care to some degree."

SOCIAL SECURITY—A GIFT HORSE?

Social Security is in trouble. Benefit funds are rapidly depleting as payments exceed federal estimates. By 1959 expenditures in excess of income may be anticipated. Deficits will be covered by higher taxes.

Rates of employer-employe Social Security participation are expected to more than double by 1975, from the two per cent contributions in 1956 to four and one-quarter per cent by 1975.

Rates for self-employed participants will increase to six and three-eighths by 1975. Currently the rate is three and three-eighths per cent.

The following table indicates the anticipated Social Security rate increase based on $4,200 annual wage.

Calendar Year	*Employe*	*Employer*	*Self-Employed*
1956	2 %	2 %	3 %
1957-59	2¼%	2¼%	3⅜%
1960-64	2¾%	2¾%	4⅛%
1965-69	3¼%	3¼%	4⅞%
1970-74	3¾%	3¾%	5⅝%
1975 and after	4¼%	4¼%	6⅜%

SHARP RISE NOTED IN HOSPITAL COSTS

The *Monthly Labor Review,* in a report on medical care costs, in the cost of living index, notes that in the past twenty years hospital costs have risen sharply in contrast to physicians' fees. The Bureau of Labor Statistics lists these increases between 1936 and 1956:

Hospital room rates	264.8%
Dentists' fees	82.1%
General Practitioners' fees	72.8%
Surgeons' fees	59.5%

In the same period, medical care costs generally have lagged behind costs of food, personal care other than medical and clothing. The report makes this observation: "With the higher level of living attained in 1950, relative expenditures for medical care tended to increase as incomes increased, as is usually true of items considered as 'necessities' in the family budget. The fact that this pattern has begun to appear in the spending of workers' families indicates the high order of importance they place on medical care. . . ."

THE NEW VA ADMINISTRATOR

Sumner G. Whittier, a former lieutenant governor of Massachusetts and an official of the Veterans Administration since last January, has been appointed chief of the agency, succeeding Harvey V. Higley who resigned recently. Mr. Whittier has been VA's chief insurance director since last January; that department handles policies covering over 6 million veterans. A Republican, Mr. Whittier was elected to his first post in 1938 as member of the Everett (Mass.) Common Council, three years after graduating from Boston University. He served in the state legislature, was in the Navy during World War II and was lieutenant governor from 1953-1956.

MEDICARE PATIENTS NOW REQUIRE IDENTIFICATION CARDS

All dependents of service personnel, with minor exceptions, are now required to present uniformed services identification and privilege card when they apply for medical service. The form number is DD Form 1173 and a photograph of the dependent appears on the reverse side. Exceptions to strict enforcement are as follows:

1. In cases of acute medical, surgical or emotional conditions of an emergency nature requiring immediate medical care for humanitarian reasons.

2. In cases of children under ten years of age. Cards have not been issued to these children and eligibility for care must be established by parents or guardian to the satisfaction of the physician.

3. In cases where, for special reasons connected with turnover of service personnel, the dependent does not possess a card. In such a case, according to the government bulletin, it will be necessary to rely on some other means of identification. It is obviously anticipated that most eligible dependents over ten years of age will be supplied with the required card.

In all cases other than those involving children under ten, the dependent or service sponsor should be required by the physician to show on the physician's claim form, DA Form 1863, in items 6 and 7 (or on an attached statement) the type of documentation or identification form used.

Only wives, dependent husbands and legitimate children of uniformed service members are eligible for care under the program.

HOW AMA DOLLARS ARE SPENT

At a recent meeting in Miami, Dr. George F. Lull, AMA secretary-general manager, told an audience how the AMA spends its annual income of approximately $10 million. The AMA's main sources of revenue are from dues and subscriptions which amount to $5,192,000, and from advertising which brings in excess of $4 million. Some expenses reported to the nearest thousand are: public relations, $400,000; Council on Medical Education and Hospitals, $376,000; Bureau of Health Education, $296,000; Washington Office, $227,000; Council on Medical Service, $206,000; Membership Records, $202,000; Bureau of Medical Economic Research, $173,000; Biographical Records, $155,000; American Medical Education Foundation Overhead, $119,000; Law Department, $111,000; Bureau of Exhibits, $109,000; Council on Drugs, $99,000; Council on Industrial Health, $76,000; Council on Rural Health, $68,000; Council on Medical Physics, $61,000, Council on Foods and Nutrition, $56,000.

THE MEDICAL PROFESSION PLAYS IMPORTANT ROLE IN 85TH CONGRESS

Numerous issues of interest and importance to the medical profession are set for action during the second session of the 85th Congress.

The Jenkins-Keogh pension plan bill (HR 9-10) is scheduled for late January hearings. Senator William Proxmire (D., Wis.) will introduce a bill embracing many features of Forand bill (HR 9467), including free hospitalization for social security recipients. Medical training is expected to receive considerable attention.

Congress is also expected to examine Medicare and decide whether any legislative changes in this program of medical services for military dependents are indicated. Also docketed for later consideration is the question of providing medical and hospitalization insurance for Federal employes and their dependents.

Expenditures for Hill-Burton hospital expansion, financial aid for medical research and contributions in support of vocational and medical rehabilitation of the physically handicapped will be included in public hearings by House Government Operations Subcommittee on Intergovernmental Relations into Federal grants for states and municipalities.

Also expected in the session, is the report and recommendations of the House Government Operations Subcommittee on Legal and Monetary Affairs regarding advertising controls for weight-reducing drugs and filter cigarettes.

SEARS-ROEBUCK FOUNDATION OFFERS AID IN DOCTOR PLACEMENTS

The Sears-Roebuck Foundation has announced a program aimed at helping communities who have no doctor to obtain the services of a doctor. The new program will stress the building or remodeling of a medical center that is either better or equal to those available in the city. Ever since the physician loan program had to be dropped, the Foundation has been working on another project in the field of medical distribution.

The Foundation has arranged with an architect to provide plans for an economical and functional medical center which is adaptable to local building materials. This building incorporates every modern feature necessary for quality medicine. It is furthermore designed to simplify the actual work load of the help by an efficient office arrangement.

The Medical center is designed to provide out-patient service where needed and can be easily expanded from a one to a two doctor unit. The cost of this building fully equipped except for examining tables, x-ray, and waiting room chairs ranges from $12,000 to $25,000. The Foundation will also be prepared to provide architectural advice on remodeling if the building is attractive and suitable.

The Foundation is prepared to provide any community so chosen to participate in this program the following: 1. Assist in conducting an economic survey of community to see if area can financially support a doctor. 2. Consultation and advice on fund raising and organizing the community. 3. Complete blueprints and building specifications on the medical center or advise on remodeling depending on which is most feasible. 4. Our services along with the AMA and our Medical Advisory Board to aid in obtaining a doctor.

Communities chosen to participate must be approved by the state society and be able to support a physician. A major consideration is whether a community is willing to help itself and attempt to raise it's own funds. Any community willing to do so regardless of its proximity to areas or medical need would qualify under this program.

If you should desire any additional information, write to Mr. Norman H. Davis, Director, The Sears-Roebuck Foundation, 3333 Arthington St., Chicago, Illinois.

U. S. EXPECTS PHYSICIAN-DENTIST SUITS

Recently the U. S. Court of Claims ruled in favor of an ex-Army dentist who, having served involuntarily as a private in 1953-54, was suing for back pay of a Dental Corps captain. Unanimous decision was that the Army flouted the doctor-draft law by using the man in his professional role while keeping him on private's pay. Court held that the military was within its rights in denying him a commission because of Communistic affiliations but erred in giving him officer's responsibility.

(Continued on Page A-57)

Medical-Legal Opinions

By JULE M. HANNAFORD, Legal Counsel
Minnesota State Medical Association

CHANGE MADE IN INDIVIDUAL INCOME TAX FORMS

Late in October, 1957, the Internal Revenue Service announced that a new line had been included in Form 1040, which is the form prepared for use by individual taxpayers in reporting their income taxes. Line 6(a) requires the listing of "Travel, reimbursed expenses, etc.," which amount is subtracted from income reported in line 5. The Instructions, which are prepared by the Internal Revenue Service for use in connection with Form 1040, explain that an employe should include in line 5 all payments he receives as reimbursement for expenses he has incurred and should deduct the expenses listed in line 6(a).

Most physicians in preparing and filing their income tax returns are in no way affected by this change. The change applies only to expenses incurred and reimbursements received by *employes.* Physicians in private practice should report the earnings and expenses of their practice in Schedule C (Form 1040) while the income and expenses of a medical partnership must be reported in Form 1065. However, some physicians receive income from their medical services in part or in whole as employes, such as physicians employed by a medical association or clinic. A physician whose income taxes are withheld from his salary and who receives a W-2 statement from his employer at the end of a year receives income as an *employe.* The change in Form 1040 would affect the preparation of tax returns by such physicians.

While physicians who are in private practice and are not employes of a medical clinic or association need not be concerned with the change made in Form 1040, a similar problem exists in determining what types of payments must be included by an individual practitioner in line 1 of Schedule C (Form 1040) as "Total receipt," or by a medical partnership in line 1 of Form 1065 as "Gross receipts." The Instructions printed on the back of Schedule C (Form 1040) state:

> "Line 1—Total Receipts.—Include all income derived from your trade or business."

Physicians on occasion bill patients for certain expenses in addition to their normal fees. For instance, a physician may find it desirable to bill a patient separately for travel, lodging, and meal expenses which are incurred in connection with services performed outside of the physician's community. Such expenses may be separately billed or shown as a separate item on a fee statement. If a physician charges a *lump sum* to cover such expenses, which is not based on the exact expenses incurred, he must include the payment he receives in his "Total receipts" ("Gross receipts" in the case of medical partnerships), and deduct the expenses which are actually incurred. On the other hand, if a physician bills a patient for the exact amount of the expenses which he has incurred and subsequently receives *reimbursement* therefor, he need not include such reimbursements in his "Total receipts." This follows both because such payments have not been made to a physician by his employer and because payments received as reimbursements of such expenses may be considered as repayments of a loan rather than as income. The same result applies to *advancements* received by a physician from a patient to cover business expenses to be incurred by the physician, where any amounts not spent for such expenses are returned or credited against the patient's bill. Of course, if in either of such instances a physician does not include the payments in his gross income, he cannot deduct the expenses. No guaranty can be made that in the future the Internal Revenue Service will not require advancements and specific reimbursements to be included in "Total receipts."

To avoid the necessity of reporting as income in tax returns payments of expenses received from patients and then detailing in tax returns the actual expenses, physicians may find it desirable to bill a patient for the exact amount of certain expenses, rather than to bill the patient an amount as an expense allowance in estimation of the expenses or to increase the bill of the patient to cover the expenses. However, the fact that such payments do not have to be reported does not mean that complete records showing the incurrence and allowability of the expenses should not be kept. If a physician's tax return should be audited, he might be requested by the Examining Revenue Agent to produce his bank statements. The Agent may ask why certain deposits were not included in the physician's income tax returns. At that time it will not be enough for the physician to state that the payments were received as reimbursements or advances for expenses; the physician will have to be able to back up his statements with proof. For that reason all physicians should strive to keep complete and detailed records. These records should itemize separately the expenses for which the physician is reimbursed, such as hotel bills, meals, train or plane fare, etc., and the reason the expenses were incurred. In addition, documentary evidence of the larger expenditures should be saved, such as ticket stubs, receipts for hotel bills, etc. Only by following such a procedure is a physician completely protected if his returns are audited.

Committee Action

The Child Health Committee

National PTA Medical Care
American Public Health Association

In May of 1956 the National Congress of Parents and Teachers adopted a series of recommendations entitled "The Periodic Health Appraisal for Well Children from Birth Through High School." Dr. Henry Helmholz, formerly of the Mayo Clinic, and a past president of the American Academy of Pediatrics, is health chairman for the National P.T.A. He was assisted in the development of these recommendations by representatives of about twenty different organizations including the American Academy of General Practice, the American Academy of Pediatrics, the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American Dental Association and the American Psychiatric Association.

This program was developed as a logical extension of the so-called "Summer Round-Up" which the P.T.A. has sponsored since 1925. The Summer Round-Up is the familiar program of health examination for children about to enter school for the first time. The new program calls for continued health supervision for all children. The groups involved in formulating this program repeatedly emphasize that the continued health supervision should be done where possible by the physician and dentist who normally serve the child and family.

National P.T.A. Recommendations

1. That the National Congress of Parents and Teachers adopt a policy supporting and encouraging a program of continuous health supervision of children from birth through their school experience, rather than only a program of single appraisal on school entrance.

2. That the National Congress of Parents and Teachers recommend to its local units a promotional and educational program that will tend to bring children and their parents into effective contact with the health resources of the community.

3. That wherever possible, these should be the physician and dentist who normally serve that child or family, whether they be working in private practice or in a community health service.

A program of continuous health supervision has been recommended repeatedly by medical, dental, school, and public health personnel. The role of the parent-teacher asociation will be to acquaint parents with the means of utilizing effectively the resources available to meet the health needs of children, beginning at birth and continuing through the school years. The parent-teacher association also should work with the professions and agencies of the community in planning and carrying out such a program.

A.P.H.A. Recommendations

A program of the character contemplated has been outlined by a committee of the American Public Health Association, in a report entitled Health Supervision of Young Children, the essential elements of which are as follows:

A. Periodic health appraisal of the child, preferably by his own physician and dentist, to give long-term continuity, including—
    1. Well-recorded medical, dental, and developmental history
    2. Physical examination to evaluate physical, mental, and emotional development and adjustment, and to discover deviations

B. Immunizations

C. Consultation with parents
    1. To advise about feeding, nutritional needs, immunizations, recommended corrective procedures, accident prevention, and health protection.
    2. To interpret what a child is like—his normal growth and his particular state of development
    3. To assist in the management and prevention of behavior and personality problems
    4. To plan required treatment and to refer parents to appropriate services when necessary
    5. To help parents become more capable and self-confident, and to build good parent-child relations and promote family well-being

D. That the National Congress of Parents and Teachers recommend to its local units that the following activities, among others, be accepted as their role in the child health program
    1. *Educational*
        (a) Acquaint residents of local communities with existing health facilities and explain how to use them effectively
        (b) Interest school personnel in the importance of preschool health as preparation for school attendance
        (c) Aid in bringing about realization of the importance of the public health unit, especially the nurse, in health promotion

(Continued on Page A-42)

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota

F. H. Magney, M.D.
Secretary

PHYSICIANS LICENSED FEBRUARY 13, 1957

January, 1957, Examination

Name	*School*		*Address*
BERZINA, Ligita Gina	U. of Minnesota	MD 1955	1800 S. Virginia St., Hopkinsville, Ky.
BROWN, Russell Thomas	Marquette U.	MD 1956	Browerville, Minn.
CHARTIER, Yvon	U. of Montreal	MD 1954	200 1st St. S.W., Rochester, Minn.
CLIMIE, Andrew Robert Wilson	U. of Glasgow, Scotland	MB 1950	200 1st St. S.W., Rochester, Minn.
GRIFFIN, Arthur Russell	U. of Wash., Seattle	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
GUENTHER, Dean Edward	U. of Wash., Seattle	MD 1956	42 E. Orme, West St. Paul, Minn.
HAYWARD. Mark Alan	U. of Manitoba	MD 1952	Gackle, N. D.
HENRIE, Joyce Elaine Rich	U. of Utah	MD 1955	200 1st St. S.W., Rochester, Minn.
JOHNSRUDE, Irwin Stanley	U. of Manitoba	MD 1956	Fairmount, No. Dak.
KING, Harold E.	U. of Wash., Seattle	MD 1955	200 1st St. S.W., Rochester, Minn.
KOLARS, Charles Paul	St. Louis Univ.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
LAPP, Maurice M.	Northwestern U.	MD 1956	Miller Hospital, St. Paul, Minn.
LOGOTHETIS, John Achilleas	U. of Thessaloniki, Greece	MD 1950	U. of Minn. Hospitals, Minneapolis, Minn.
LU, Cheng-en	West China Union U., Cheng-tu, China	MD 1949	3312 First Ave. S., Minneapolis 8, Minn.
MIGAKI, Hoyoko	U. of Oregon	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
MOLLER, Jurgen	Christian-Albrechts U., Kiel, Germany	MD 1952	331 Oak St., Farmington, Minn.
MORAN, Walter Harrison	Harvard U.	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
MULHAUSEN, Robert Oscar	U. of Illinois	MD 1955	Vet. Adm. Hospital, Minneapolis, Minn.
MUNRO, Jerrold Albert	U. of Minnesota	MD 1955	4719 Townes Road, Edina, Minn.
NORSTROM, Craig Wilbur	U. of Alberta	MD 1955	200 1st St. S.W., Rochester, Minn.
PASH, Earl Leon	U. of Manitoba	MD 1950	U. of Minn. Hospitals, Minneapolis, Minn.
READ, Raymond Charles	U. of Minnesota	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
SCOTT, Alan Brown	U. of California	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
SMITH, Jr., Lester Leigh	Med. Col. of S. Car.	MD 1953	200 1st St. S.W., Rochester, Minn.
THAL, Alan Philip	U. of Cape Town, S. Africa	MB, Ch.B. 1949	U. of Minn. Hospitals, Minneapolis, Minn.
TROTMAN, Neil McKay	U. of Illinois	MD 1956	1698 Ford Pkwy., St. Paul 16, Minn.
UTZ, Philip Hoy	U. of Michigan	MD 1953	La Crescent, Minn.
WEINSTEIN, Marvin Jerome	U. of Oregon	MD 1955	200 1st St. S.W., Rochester, Minn.
WISE, James Kearney	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
BACHHUBER, Max Otto	U. of Wisconsin	MD 1932	Alma, Wis.
BARRIER, Jr., Charles Wesley	U. of Texas	MD 1954	200 1st St. S.W., Rochester, Minn.
BRUNGARDT, Bernard Aloysius	Creighton U.	MD 1946	200 1st St. S.W., Rochester, Minn.
ENGSTROM, Jr., Perry H.	U. of Pittsburgh	MD 1947	403 7th St. N., Wahpeton, No. Dak.
GILES, Jr., William Fisher	Creighton U.	MD 1955	200 1st St. S.W., Rochester, Minn.
HARRIS, James Roe	U. of Wisconsin	MD 1951	Gen. Delivery, Winston-Salem, N. C.
HOLBERT, James Allen	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.
JACKSON, J. Albert	U. of Pittsburgh	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
KEEFE, William Peter	U. of Maryland	MD 1955	200 1st St. S.W., Rochester, Minn.
LARSON, Norman Eugene	Northwestern U.	MD 1953	200 1st St. S.W., Rochester, Minn.
LINKE, Charles Allen	U. of Illinois	MD 1950	205 W. 2nd St., Duluth, Minn.
McALLISTER, James	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.
McGOON, Dwight Charles	Johns Hopkins U.	MD 1948	200 1st St. S.W., Rochester, Minn.
MITCHELL, William Charles	U. of Louisville	MD 1952	200 1st St. S.W., Rochester, Minn.
NEIL, Robert Lewis	U. of Tennessee	MD 1951	200 1st St. S.W., Rochester, Minn.
NEUMANN, Jr., Roland Ferdinand	Washington U., St. Louis, Mo.	MD 1944	1935 Med. Arts Bldg., Minneapolis 2, Minn.
NIEHAUS, Karl Friedrich	U. of Nebraska	MD 1953	200 1st St. S.W., Rochester, Minn.
PETERSEN, Byron Douglas	U. of Nebraska	MD 1947	U. of Minn. Hospitals, Minneapolis, Minn.
PORTELA-MORALES, Angel Ismael	Med. Col. of Va.	MD 1951	200 1st St. S.W., Rochester, Minn.
REICHELDERFER, Thomas Elmer	Johns Hopkins U.	MD 1950	Rm. 11N322, Bldg. 10, Nat. Inst. of Health, Bethesda 14, Md.
RICE, William Henry	U. of Nebraska	MD 1956	825 S. Oak St., Owatonna, Minn.

Name	*School*			*Address*
ROBERTS, George Albert	U. of Illinois	MD	1944	St. Olaf's Hospital, Austin, Minn.
SMITH, John Hough	U. of Michigan	MD	1955	200 1st St. S.W., Rochester, Minn.
WARVEL, Jr., John Henry	Johns Hopkins U.	MD	1955	200 1st St. S.W., Rochester, Minn.
WEBB, Jr., Robert Basye	U. of Virginia	MD	1952	200 1st St. S.W., Rochester, Minn.
WEISSMAN, Fredrick	Wayne Univ.	MD	1953	200 1st St. S.W., Rochester, Minn.
WILLIAMS, Hugh Jones	U. of West Ontario	MD	1950	200 1st St. S.W., Rochester, Minn.

NATIONAL BOARD CANDIDATES

Name	*School*			*Address*
ADDISON, Robert Meade	Northwestern U.	MD	1953	200 1st St. S.W., Rochester, Minn.
BOYD, Frank Elmer	Harvard U.	MD	1953	Jasper, Minn.
ENGEL, Andrew George	McGill Univ.	MD	1955	Nat. Cancer Inst., Nat. Inst. of Health, Bethesda, Md.
FRANCKOWIAK, John Joseph	Georgetown U.	MD	1955	200 1st St. S.W., Rochester, Minn.
HAMILTON, Michael Joseph	Creighton U.	MD	1952	200 1st St. S.W., Rochester, Minn.
JARCHOW, Brian H.	Loyola U.	MD	1953	200 1st St. S.W., Rochester, Minn.
LARSON, Robert LeRoy	Geo. Washington U., Washington, D. C.	MD	1953	200 1st St. S.W., Rochester, Minn.
OKIHIRO, Michael Masaru	U. of Michigan	MD	1955	200 1st St. S.W., Rochester, Minn.
ROSS, Helen Margaret	Northwestern U.	MD	1955	200 1st St. S.W., Rochester, Minn.
STERLING, Harold Melvin	Yale Univ.	MD	1951	U. of Minn. Hospitals, Minneapolis, Minn.
TASWELL, Howard Filmore	New York Univ.	MD	1953	200 1st St. S.W., Rochester, Minn.
WILBUR, Jr., Oscar Milton	Boston Univ.	MD	1946	Hibbing Gen. Hospital, Hibbing, Minn.

PHYSICIANS LICENSED MAY 24, 1957

April, 1957, Examination

Name	*School*			*Address*
BERGER, Saul	U. of Toronto	MD	1943	901 Boyd Bldg., Winnipeg, Man., Can.
BLEDSOE, Francis Henry	Med. Col. of S.C.	MD	1953	200 1st St. S.W., Rochester, Minn.
CAMPBELL, Mervin Willard	Northwestern U.	MD	1956	8821 Jenrich Ave., Midway City, Cal.
DAVIDSON, William David	Duke Univ.	MD	1953	St. Joseph, Minn.
ENGLAND, Rodney Wayne	U. of Illinois	MD	1956	1714 E. Brown St., Springfield, Ill.
FRANK, Gilbert Sherman	U. of Minnesota	MD	1954	1807 Kentucky Ave., St. Louis Park, Minn.
HATHAWAY, Walter Ryland	Marquette U.	MD	1956	531 N. Main St., South Bend, Ind.
HEINZ, John N.	Loyola U.	MD	1956	109 Lincoln Ave. S.W., Wadena, Minn.
HO, Shu Kang	Army Med. College, Chekiang, China	MB	1947	1700 University Ave., St. Paul 4, Minn.
JACKSON, Carl Robert	Jefferson Med. Col.	MD	1956	1027 E. Johnson St., Madison, Wis.
JUDGE, Crawford MacCullough	U. of Manitoba	MD	1951	5584 Harvard Place, Ontario, Cal.
LEES, Jack Ramon	Northwestern U.	MD	1956	1328 2nd So., Fargo, N. D.
MUHICH, Donald Francis	Harvard U.	MD	1956	513 Grant Ave., Eveleth, Minn.
PETERSON, John Robert	U. of Oregon	MD	1956	Wisdom, Montana
SNYDER, Joseph	U. of Wash., Seattle	MD	1955	301 W. 37th St., Topeka, Kansas
TAYLOR, Gordon Chris	Washington U. St. Louis, Mo.	MD	1956	U. of Minn. Hospitals, Minneapolis, Minn.
WHITE, James Edwin	U. of Oklahoma	MD	1954	Vet. Adm. Hospital, Minneapolis, Minn.
YOUNG, Hadley Rasch	Duke Univ.	MD	1956	929 Med. Arts Bldg., Duluth 2, Minn.
ZUPAN, George	U. of Oregon	MD	1956	4036 S. 6th St., Klamath Falls, Ore.

RECIPROCITY CANDIDATES

Name	*School*			*Address*
AIGNER, Bruno Robert	St. Louis U.	MD	1952	200 1st St. S.W., Rochester, Minn.
EVENSON, David Johann	Stanford U.	MD	1954	200 1st St. S.W., Rochester, Minn.
GATZKE, Laurence Duane	U. of Iowa	MD	1955	200 1st St. S.W., Rochester, Minn.
GRISMER, Jerome Theodore	U. of Cincinnati	MD	1948	4959 Excelsior Blvd., Minneapolis 16, Minn.
HANSKE, Edward Albert	U. of Iowa	MD	1947	402 Phys. & Surg. Bldg., Minneapolis Minn.
KOLP, Berton Andrew	U. of Texas	MD	1950	105 E. Minn. Ave., Glenwood, Minn.
LEPPINK, Harold Bernard	Wayne Univ.	MD	1949	Two Harbors, Minn.
LIEBER, Arthur	U. of Louisville	MD	1953	Vet. Adm. Hospital, Minneapolis, Minn.
OTTO, Evan Louis	Northwestern U.	MD	1951	Vet. Adm. Hospital, Vancouver, Wash.
PERSON, Douglas Allen	U. of Michigan	MD	1955	204 Med. Arts Bldg., Albert Lea, Minn.
REED, Henry Hofmann	U. of Kansas	MD	1953	205 W. 2nd St., Duluth, Minn.
REITER, Benjamin Reynolds	Harvard U.	MD	1934	U. of Minn., Stud. H.S., Minneapolis, Minn.
ROSS, Jr., James Vincent	Duke Univ.	MD	1955	200 1st St. S.W., Rochester, Minn.
SUMMAR, Marion Thomas	Tulane Univ.	MD	1949	727 Grant Ave., Virginia, Minn.
SWANSON, Wallace Leroy	Bowman Gray Univ.	MD	1956	3017 Bloomington Av. S., Minneapolis, Minn.
TAMA, Lawrence	U. of Penna.	MD	1953	200 1st St. S.W., Rochester, Minn.
WILBUR, III, Dwight Locke	Stanford U.	MD	1955	200 1st St. S.W., Rochester, Minn.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
BOOLUKOS, George Peter Nicholas	Albany Med. Col.	MD 1953	200 1st St. S.W., Rochester, Minn.
BRADLEY, John Grover	Geo. Wash. U.	MD 1951	629 Med. Arts Bldg., Minneapolis 2, Minn.
FRIEDLIEB, Oskar Peter	American U. of Beirut	MD 1948	2215 Plymouth Ave. N., Minneapolis, Minn.
MOLLER, Julia Clara	Med. Col. of Va.	MD 1953	1823 E. 5th St., Duluth, Minn.
PETERSON, Clifton Edward	U. of Rochester	MD 1954	200 1st St. S.W., Rochester, Minn.
STEVENS, William Augustus	New York Med. Col.	MD 1955	Vet. Adm. Hospital, Minneapolis, Minn.

PHYSICIANS LICENSED JULY 12, 1957

June, 1957, Examination

Name	*School*		*Address*
ANDERSON, Allen Merle	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
ANDERSON, Daniel Clarence	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
BAAK, William W.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
BACH, Jerome Menuhim	U. of Minnesota	MD 1957	USPHS Hospital, Baltimore, Md.
BALIAN, Lucy June	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
BARNARD, Jr., George Linal	U. of Minnesota	MD 1953	U.C.L.A. Med. Center, Los Angeles, Cal.
BAUDER, George B.	U. of Minnesota	MD 1957	Asbury Meth. Hospital, Minneapolis, Minn.
BEALKA, Richard James	St. Louis U.	MD 1956	St. Joseph's Hospital, St. Paul, Minn.
BEGGS, John Harkness	U. of Minnesota	MD 1957	Gorgas Hospital, Ancon, Canal Zone
BELAU, Paul Garnett	U. of Minnesota	MD 1957	USPHS Hospital, Staten Island, N. Y.
BEN-ORA, Avi	U. of Minnesota	MD 1957	San Joaquin Gen. Hosp., French Camp, Cal.
BERENDES, Heinz	U. of Munich, Germany	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
BERGSTROM, LaVonne B.	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis, Minn.
BERMAN, John Alexander	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
BINGHAM, George Conley	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
BRADLEY, William Edward	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
BRENK, Paul E.	U. of Minnesota	MD 1957	U. S. Army Hospital, Fort Benning, Ga.
BROOKS, Bancroft Morgan	U. of Minnesota	MD 1957	Kaiser Fdtn. Hospital, San Francisco, Cal.
BROWN, Charles Talcott	U. of Minnesota	MD 1957	Bellevue Hospital, New York, N. Y.
BROWN, Frank Thomas	U. of Minnesota	MD 1957	Parkland Mem. Hospital, Dallas, Texas
BROWN, Hubert Marion	Johns Hopkins U.	MD 1953	U. of M. Hosp., Dept. Derm., Mpls, Minn.
CADMAN, Norman LeRoy	U. of Chicago	MD 1953	200 1st St. S.W., Rochester, Minn.
CALVERLEY, John Robert	U. of Oregon	MD 1955	200 1st St. S.W., Rochester, Minn.
CASEY, John J.	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
CHAPMAN, George Robert	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis, Minn.
CHRISTENSON, Leland Roger	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
CROW, George Michael	U. of Minnesota	MD 1957	Mercy Hospital, Toledo, Ohio
CUNNINGHAM, Richard Donald	U. of Minnesota	MD 1957	Tripler Army Hospital, Honolulu, Hawaii
DAHL, Diane Anita	U. of Wisconsin	MD 1956	814 Mahtomedi Ave., Mahtomedi, Minn.
DALY, Alfred Emanuel	St. Louis U.	MD 1957	Tripler Army Hospital, Honolulu, Hawaii
DAWSON, Jr., Walter John	U. of Minnesota	MD 1957	St. Luke's Hospital, Cleveland, Ohio
DIVERTIE, Matthew Burgess	U. of Glasgow, Scotland	(Bach. Med. & Surg.) 1947	200 1st St. S.W., Rochester, Minn.
DUDLEY, James Patrick	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
DVORAK, Jr., Benjamin Anthony	Vanderbilt U.	MD 1957	Vanderbilt U. Hospital, Nashville, Tenn.
EHRLICH, Jr., S. Paul	U. of Minnesota	MD 1957	USPHS Hospital, Staten Island, N. Y.
ENGSTROM, E. Duane	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
EUSTERMAN, Joseph Huntimer	U. of Minnesota	MD 1957	Abington Mem. Hospital, Abington, Pa.
FAIRBANKS, Leland L.	U. of Minnesota	MD 1957	USPHS Hospital, New Orleans, La.
FEHR, Peter Eilert	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
FOLEY, Robert Russell	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose, Cal.
GELLER, Joseph	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
GIESBRECHT, Arnold	U. of Manitoba	MD 1957	Hallock, Minn.
GLETNE, John Sanford	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose, Cal.
GOLDFINE, M. Melvin	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
GOLDSTEIN, Alan Lawrence	U. of Minnesota	MD 1957	St. Charles Hospital, Toledo, Ohio
GONIOR, Thomas Henry	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
GOOD, Gary Daniel	U. of Minnesota	MD 1957	Salt Lake Co. Gen. Hosp., Salt Lake City, U.
GOODMAN, Ernest	U. of Bologna, Italy	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
GOODMAN, Malka Lotterstein	U. of Minnesota	MD 1956	1053 Iglehart Ave., St. Paul, Minn.
GRAHAM, Asa Briggs	U. of Minnesota	MD 1957	Mercy Hospital, Toledo, Ohio
HANEY, David George	U. of Minnesota	MD 1957	Wayne Co. Gen. Hosp., Eloise, Mich.
HARDER, Donald S.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
HARTY, Jerome Leo	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
HEGGESTAD, Carl Blixseth	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
HILGER, John Rothrock	McGill U.	MD 1957	Ancker Hospital, St. Paul, Minn.
HILKER, Robert Tolan	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
HOLDCRAFT, John Willard	Jefferson Med. Col.	MD 1956	307 Myrtle Ave., Woodbury, N. J.
HORNS, Norman Mather	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
HOVERSTEN, Vincent Wordsworth	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose 28, Cal.

Name	*School*		*Address*
JACKISH, George Edward	U. of Minnesota	MD 1957	Hurley Hospital, Flint, Mich.
JAEGER, Dwight E.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
JOHNSON, Doris I.	U. of Minnesota	MD 1957	Orange Co. Hospital, Orange, Cal.
JOHNSON, Wendell August	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
JOHNSON, William Stark	U. of Illinois	MD 1952	200 1st St. S.W., Rochester, Minn.
KAYUTE, Sheldon Wayne	U. of Minnesota	MD 1949	State Hospital, Willmar, Minn.
KEAIRNES, Harold Wrenn	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
KOZAK, Michael John	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
KVISTBERG, Gerald Kent	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
KYLLONEN, Ronald Rudolph	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
LARSON, Dean Weston	U. of Minnesota	MD 1957	City Hospital, Cleveland, Ohio
LICHTIG, Moses	U. of Minnesota	MD 1957	Montefiore Hospital, New York, N. Y.
LIFSON, William L.	U. of Minnesota	MD 1957	Milw. Co. Hospital, Milwaukee, Wis.
LINDHOLM, Dale David	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
LUND, Richard Roy	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
LYNCH, Richard Patrick	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
LYON, Fred Alexander	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
MARK, Merle Stanley	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MATSON, Roland Willatz	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
McGUIRE, Arthur Merritt	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
MENA, Abelardo	National Univ. of South East, Mexico	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
MLINAR, Joseph Paul	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
MODELL, Jerome Herbert	U. of Minnesota	MD 1957	St. Albans Naval Hosp., St. Albans, L. Is., New York
MOE, Walter Wyatt	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
MORI, Hideo	U. of Chicago	MD 1956	924 Essex St. S.E., Minneapolis, Minn.
MUCHOW, Gene Carleton	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MULVAHILL, John Edward	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MUSA, Byron Ulysses	U. of Oregon	MD 1956	708 W. Deschutes, Redmond, Ore.
MUZZALL, Hugh Arthur	U. of Wash., Seattle	MD 1956	206 E. 10th, Ellensburg, Wash.
NADEAU, Pierre	U. of Montreal	MD 1954	200 1st St. S.W., Rochester, Minn.
NAROS, Jack Albert	U. of Minnesota	MD 1957	So. Pacific Gen. Hosp., San Francisco, Cal.
NIAZI, Suad Asad	Royal Col. Med., Baghdad, Iraq	Bach. Med. & Surg. 1941	Box 446, Clarkfield, Minn.
NIELSEN, Andrew Adolf	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
NILSEN, John Anton	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
OLSON, Robert Joseph	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
OPHEIM, Richard Henry	U. of Minnesota	MD 1957	So. Pacific Gen. Hosp., San Francisco, Cal.
ORBUCH, Martin Walter	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
OSE, Anna Leonija	U. of Latvia, Riga, Latvia	"Phys." 1931	State School & Hospital, Cambridge, Minn.
OSS, Ausma Velta	U. of Minnesota	MD 1957	St. Michael Hospital, Newark, N. J.
PAWLYSZYN, Julian	U. of Heidelberg, Germany	MD 1949	State Hospital, Anoka, Minn.
PETERSON, Charles Allan	U. of Minnesota	MD 1957	USPHS Hospital, Boston, Mass.
PETERSON, John Alfred	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
POBOR, Paul H.	U. of Minnesota	MD 1957	Los Angeles Co. Hosp., Los Angeles, Cal.
POLLARD, John William	U. of Minnesota	MD 1957	Valley Forge Army Hosp., Phoenixville, Pa.
REEMSNYDER, Curtis C.	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
ROACH, Charles Albert	U. of Minnesota	MD 1957	Miller Hospital, St. Paul, Minn.
ROBINSON, James Parker	U. of Minnesota	MD 1957	Walter Reed Army Hospital, Wash., D. C.
RUSHAY, Arthur James	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
RYDBERG, John S.	U. of Minnesota	MD 1957	Receiving Hospital, Detroit, Mich.
SACKS, Jerome Henry	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
SAYTHER, Keith Duddley	U. of Minnesota	MD 1957	San Joaquin Co. Hosp., French Camp, Cal.
SCHEREK, Jerome John	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SCHLOFF, Ivan	U. of Bologna, Italy	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
SCHROEPFER, Jr., George John	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
SHAPIRO, Stanley William	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
SHEFVELAND, John Ronald	U. of Minnesota	MD 1957	Harbor Gen. Hospital, Torrance, Cal.
SHUMRICK, Donald Albert	U. of Minnesota	MD 1957	San Fran. Cty.-Co. Hosp., San Fran., Cal.
SINGBEIL, Julius Henry	U. of Manitoba	MD 1956	123 N. 6th St., Breckenridge, Minn.
SMITH, Henry Nippert	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SOLLIE, Stanley Carol	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
SPRAITZ, Jr., Anton Francis	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SUTHER, Lennart Elmer	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
TANQUIST, Jr., Edwin John	U. of Minnesota	MD 1957	Gorgas Hospital, Ancon, Canal Zone
THEOBALT, Inge Marie Bartsch	U. of Kiel, Germany	MD 1944	U. of Minn. Hospitals, Minneapolis, Minn.
THOMPSON, Wayne William	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
TOUSSAINT, Florence Amalia	U. of Minnesota	MD 1957	Queen's Hospital, Honolulu, Hawaii
VALGEMAE, Romil	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
VAN DE WATER, Frank W.	U. of Minnesota	MD 1957	Denver City Gen. Hosp., Denver, Colo.
VAN PUFFELEN, Paul S.	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.

Name	*School*		*Address*
WELLS, Ralph Frederick	U. of Minnesota	MD 1957	Brooke Army Hospital, San Antonio, Tex.
WILKEN, Robert Wayne	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
WILSON, George Patterson	Washington U., Mo.	MD 1956	Vet. Adm. Hospital, Minneapolis, Minn.
WORLTON, Jr., James Timbrell	U. of Utah	MD 1954	200 1st St. S.W., Rochester, Minn.
YOUNG, Joseph Orville	U. of Minnesota	MD 1957	Tripler Army Hospital. Honolulu, Hawaii
ZAK, Solomon Joseph	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
CHAPPELL, Elliott Rae	U. of Nebraska	MD 1944	112 S. Main St., Stillwater, Minn.
FALK, Abraham	Albany Med. Col.	MD 1936	2500 Como Ave., St. Paul, Minn.
FOX, Philip Martin	U. of California	MD 1947	Vet. Adm. Hospital, Minneapolis, Minn.
GERBER, Edward Paul	U. of Illinois	MD 1953	200 1st St. S.W., Rochester, Minn.
GIDDINGS, Frank Chandler	U. of Nebraska	MD 1954	Miller Hospital, St. Paul, Minn.
HUFF, James Foster	Louisiana State U.	MD 1955	200 1st St. S.W., Rochester, Minn.
LINKE, Clara Julia Leinberger	U. of Illinois	MD 1950	St. Luke's Hospital, Duluth, Minn.
McCARTHY, John Denis	U. of Rochester	MD 1955	200 1st St. S.W., Rochester, Minn.
McHUTCHISON, Samuel Kerr	U. of Pennsylvania	MD 1955	St. Charles, Minn.
SCHNABEL, Robert Frederick	Med. Col. of Va.	MD 1951	220 S. Broadway, Crookston, Minn.
WARDEN, Herbert Edgar	U. of Chicago	MD 1946	U. of Minn. Hosp., Dept. Surg., Mpls.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
ABSOLON, Karel Bedrich	Yale U.	MD 1952	Key West Naval Hosp., Key West, Fla.
ANDERSEN, James Gordon	Harvard U.	MD 1952	U. S. Vet. Adm. Hosp., Boise, Idaho
GIULIANI, Emilio R.	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
HAWK, Dale Jay	Hahnemann, Pa.	MD 1951	St. Charles, Minn.
JEWELL, Jr., Albert Hartwell	Northwestern U.	MD 1950	209 W. Mill St., Austin, Minn.
STEIDL, Richard Meredith	Albany Med. Col.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
TORO-NAZARIO, Rafael Andres	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
VASTOLA, Jr., Anthony Paul	Boston U.	MD 1953	200 1st St. S.W., Rochester, Minn.
VOGEL, Gerald William	U. of Chicago	MD 1954	200 1st St. S.W., Rochester, Minn.
WAGNER, Robert Morris	Cornell U.	MD 1952	507 Med. Arts Bldg., Minneapolis, Minn.

PHYSICIANS LICENSED NOVEMBER 8, 1957

October, 1957, Examination

Name	*School*		*Address*
CHEDISTER, Charles Russell	U. of Illinois	MD 1956	4119 2nd Ave. So., Minneapolis, Minn.
CONN, Jr., Rex Boland	Yale Univ.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
DEASON, Keith Burdette	Creighton U.	MD 1957	St. Luke's Hospital, Duluth, Minn.
DIRE, William Nick	U. of Oregon	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
ECKHARDT, Arthur Lloyd	U. of Oregon	MD 1957	Ancker Hospital, St. Paul, Minn.
EICHENHOLZ, Alfred	Ludwig-Maximilian U., Munich, Germany	MD 1951	Vet. Adm. Hospital, Minneapolis, Minn.
FLOYD, Malcolm Stafford	Med. Col. of S. Car.	MD 1956	200 1st St. S.W., Rochester, Minn.
FLUEGEL, John Ormond	Creighton U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
FORBES, Edward Francis	Creighton U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
GILBERT, Robert Alfred	Columbia U.	MD 1956	200 1st St. S.W., Rochester, Minn.
HAKALA, Edwin Wayne	U. of Minnesota	MD 1938	505 Beltzell Ave., Fort Benning, Ga.
JENKINS, Wallace Vernon	U. of Utah	MD 1955	Mpls. Gen. Hospital, Minneapolis 15, Minn.
JOHNSON, Thomas Errol	U. of Nebraska	MD 1954	Miller Hospital, St. Paul, Minn.
KNUDSEN, Jerry Milo	Baylor Univ.	MD 1956	Ancker Hospital, St. Paul, Minn.
LINDSETH, Esten Opland	U. of Oslo, Norway	MD 1949	U. of M. Grad School, Minneapolis, Minn.
McQUARRIE, Donald Gray	U. of Utah	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
O'LOUGHLIN, Peter Daniel	Marquette U.	MD 1955	St. Mary's Hospital, San Francisco, Cal.
RAKOW, Raymond William	U. of California	MD 1953	200 1st St. S.W., Rochester, Minn.
RECHT, Thomas Monroe	U. of Nebraska	MD 1956	Mpls. Gen. Hospital, Minneapolis 15, Minn.
REISCH, Alvin J.	Northwestern U.	MD 1956	200 1st St. S.W., Rochester, Minn.
REYNOLDS, James Francis	U. of Penna.	MD 1956	200 1st St. S.W., Rochester, Minn.
REYNOLDS, William Arthur	Washington U. (Mo.)	MD 1956	200 1st St. S.W., Rochester, Minn.
SCHORR, William Francis	Marquette U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
SEIFERT, Richard Henry	Loyola Univ.	MD 1957	St. Joseph's Hospital, St. Paul, Minn.
SHANAHAN, Daniel Francis	Creighton U.	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
WHITMAN, Edwin Joel	U. of Pittsburgh	MD 1956	200 1st St. S.W., Rochester, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
AHERN, Gene	U. of Illinois	MD 1953	1218 S. Park, Red Wing, Minn.
BALLANTINE, Jerome Jennings	Indiana Univ.	MD 1956	Vet. Adm. Hospital, Minneapolis, Minn.
BOLLIGER, Eugene Frederick	U. of Michigan	MD 1946	Northwestern Hospital, Minneapolis, Minn.
BRODHUN, John Charles	Marquette U.	MD 1954	915 3rd Ave. S.E., Rochester, Minn.
BROWN, Paul Marvin	Harvard U.	MD 1949	200 1st St. S.W., Rochester, Minn.

Name	*School*		*Address*
BYNUM, Jr., Grover LaFayette	Tulane U.	MD 1953	200 1st St. S.W., Rochester, Minn.
CARVETH, Stephen Walker	U. of Nebraska	MD 1956	200 1st St. S.W., Rochester, Minn.
COWGER, Marilyn Louise	U. of Nebraska	MD 1956	200 1st St. S.W., Rochester, Minn.
CULLEN, Jr., Paul Kent	U. of Indiana	MD 1956	200 1st St. S.W., Rochester, Minn.
DAW, Edward Francis	State U. of Iowa	MD 1954	200 1st St. S.W., Rochester, Minn.
DOMBROWSKI, Jr., Edmund T.	U. of California	MD 1956	200 1st St. S.W., Rochester, Minn.
GILDERSLEEVE, Robert G.	Col. Med. Evang.	MD 1948	Watertown, Minn.
GILLOON, James Robert	Creighton U.	MD 1953	200 1st St. S.W., Rochester, Minn.
GRAUDINS, Gunars	U. of Iowa	MD 1956	200 1st St. S.W., Rochester, Minn.
HEILMAN, Richard Owen	U. of Wisconsin	MD 1955	1003 Oak St., Tomah, Wis.
HENKE, Frederick William	U. of Wisconsin	MD 1945	Vet. Adm. Hospital, Minneapolis, Minn.
INLOW, Robert Pierson	Indiana U.	MD 1956	200 1st St. S.W., Rochester, Minn.
KEIG, Jr., William Paul	Northwestern U.	MD 1947	1600 University Ave., Grand Forks, N. D.
KINNARD, Jr., John Parkes	U. of Tennessee	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
KUNDEL, Robert Ray	U. of Iowa	MD 1956	208 E. Chapman, Ely, Minn.
LILLIE, Andrew Rendall	U. of Manitoba	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
LORENC, Ernest	U. of Iowa	MD 1954	200 1st St. S.W., Rochester, Minn.
McPARLAND, Jr., Felix Augustus	Western Reserve	MD 1950	4841 E. Lake Harriet Blvd., Mpls., Minn.
MILLER, Jr., Archie William	U. of Michigan	MD 1956	200 1st St. S.W., Rochester, Minn.
MUNGER, James Everett	Ohio State U.	MD 1954	8506 Beverly St., Duluth 8, Minn
NELSON, Donald Keith	Indiana U.	MD 1956	200 1st St. S.W., Rochester, Minn.
NORBECK, David Eskel	U. of Illinois	MD 1952	303 Doctors Bldg., Minneapolis, Minn.
POSEY, John William	U. of Nebraska	MD 1951	200 1st St. S.W., Rochester, Minn.
SCHEEN, Jr., Sanuel Randolph	U. of Louisville	MD 1953	200 1st St. S.W., Rochester, Minn.
STORY, Jimmy Lewis	Vanderbilt U.	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
TAYLOR, Lawrence Carol	U. of Nebraska	MD 1955	200 1st St. S.W., Rochester, Minn.
TIPPITT, Nathaniel George	Louisiana State U.	MD 1950	200 1st St. S.W., Rochester, Minn.
WILEY, Alden Francis	U. of Iowa	MD 1953	Box 176, Kalkaska, Mich.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
ADAMS, Ralph Edwin	U. of Oregon	MD 1956	200 1st St. S.W., Rochester, Minn.
ALLAMAN, Loren Ellsworth	Northwestern U.	MD 1954	200 1st St. S.W., Rochester, Minn.
BATTAILE, William George	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
BERNSTEIN, Eugene Felix	State U. of N. Y.	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
BOROSON, Hugh Norbert	New York Univ.	MD 1951	Northw'n Hosp., Thief River Falls, Minn.
BRINK, Benno Marcellus	Creighton U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CLARK, Lealand L.	Columbia U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CORR, William Philip	Stanford U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CRANDALL, Earle Ellsworth	U. of Illinois	MD 1956	200 1st St. S.W., Rochester, Minn.
DAVIDSON, Paul	Albany Med. Col.	MD 1956	200 1st St. S.W., Rochester, Minn.
DONDERO, Jr., John A.	Georgetown U.	MD 1952	58 85th St., Brooklyn 9, N. Y.
EDELSTEIN, Jack Paul	U. of Chicago	MD 1956	200 1st St. S.W., Rochester, Minn.
ENGLER, Robert Sherman	Columbia U.	MD 1954	200 1st St. S.W., Rochester, Minn.
ETZWILER, Donnell Dencil	Yale Univ.	MD 1953	445 E. 68th St., New York 21, N. Y.
FARLEY, Harrison Hatheway	Cornell Med. Col.	MD 1954	Mpls. Gen. Hospital, Minneapolis 15, Minn.
FAUST, Herbert Arlington	Hahnemann Med. Col.	MD 1952	200 1st St. S.W., Rochester, Minn.
GRABOW, Jack David	U. of Buffalo	MD 1956	200 1st St. S.W., Rochester, Minn.
HIGGINS, John Elton	Dalhousie U.	MD 1951	200 1st St. S.W., Rochester, Minn.
HOOPS, Jr., Harold John	Columbia U.	MD 1954	200 1st St. S.W., Rochester, Minn.
HOPKINS, Donald Marks	Northwestern U.	MD 1956	200 1st St. S.W., Rochester, Minn.
HUNT, James Cleon	U. of Illinois	MD 1954	200 1st St. S.W., Rochester, Minn.
KERN, Kathryn Eustance	U. of Rochester	MD 1956	4921 Miss. Court, Minneapolis, Minn.
KERN, Jr., William Albert	U. of Rochester	MD 1956	Mpls. Gen. Hospital, Minneapolis 15, Minn.
KINZEL, Raymond Christian	New York Med. Col.	MD 1955	200 1st St. S.W., Rochester, Minn.
KIRSHEN, Robert	Chicago Med. Sch.	MD 1956	200 1st St. S.W., Rochester, Minn.
MAYTUM, Wellington James	U. of Penna.	MD 1956	200 1st St. S.W., Rochester, Minn.
MELNICK, Gilbert Stanley	State U. of N. Y.	MD 1954	1338 W. Maynard Dr., St. Paul, Minn.
MURPHY, Frank Patrick	Loyola U.	MD 1954	200 1st St. S.W., Rochester, Minn.
NYDELL, Jr., Carl Clifford	Col. Med. Evang.	MD 1954	200 1st St. S.W., Rochester, Minn.
REX, James Caldwell	Temple U.	MD 1950	200 1st St. S.W., Rochester, Minn.
RICE, Bernard Francis	Temple U.	MD 1956	200 1st St. S.W., Rochester, Minn.
SHERRICK, Donald William	Yale Univ.	MD 1956	200 1st St. S.W., Rochester, Minn.
SPIEKERMAN, Ralph Earl	U. of Illinois	MD 1954	200 1st St. S.W., Rochester, Minn.
SPURGEON, Frederick Calvin	New York Med. Col.	MD 1953	200 1st St. S.W., Rochester, Minn.
THOMPSON, III, John William	St. Louis Univ.	MD 1954	200 1st St. S.W., Rochester, Minn.
WEAVER, Walt Franklin	Western Reserve	MD 1956	200 1st St. S.W., Rochester, Minn.
WEBB, Harry Edward	U. of Kansas	MD 1956	200 1st St. S.W., Rochester, Minn.
WESTLIN, Jr., William Frederick	New York Med. Col.	MD 1954	200 1st St. S.W., Rochester, Minn.
WHERRY, David Colwell	Geo. Washington U.	MD 1952	504 Monticello Blvd., Alexandria, Va.
WHITE, James Colin	U. of Vermont	MD 1956	200 1st St. S.W., Rochester, Minn.
WOELLNER, Richard Carlton	U. of Chicago	MD 1955	Sch. Aviation Med., Naval Air Station, Pensacola, Fla.
WRY, Paul Edward	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.

Meetings and Announcements

STATE

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

NATIONAL

American College of Surgeons, sectional meeting, Des Moines, Iowa, March 27-29, 1958.

American Congress of Physical Medicine and Rehabilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

Chicago Medical Society, Annual Clinical Conference, Palmer House, Chicago, Illinois, March 4, 5, 6, and 7, 1958.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

International College of Surgeons, 11th biennial International Congress, in conjunction with 23rd annual Congress of United States and Canadian Sections (North American Federation), Los Angeles, California, March 9-14, 1958.

International Society of Internal Medicine, Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy Association, Paris, France, October 19-26, 1958.

World Congress of Gastroenterology, Washington, D. C., May 25-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

ISAAC RAY LECTURE SERIES

Dr. Manfred S. Guttmacher, psychiatrist to the Supreme Bench of Baltimore, will speak Monday, Tuesday and Thursday evenings, March 3, 4 and 6, in the 1958 Isaac Ray lecture series to be at the University of Minnesota under the joint sponsorship of the University's law and medical schools.

Dr. Guttmacher will discuss "Confidentiality and Privileged Communication," "The Psychotic Murderer" and "Psychiatric Court Clinics" in his three lectures which will be presented primarily for doctors and lawyers. The lectures, which will be given in Mayo Auditorium, will begin at 8 p.m. each of the three evenings.

POSTGRADUATE COURSE

The second Annual Postgraduate Course in Fractures and Other Trauma will be given by the Chicago Committee on Trauma of the American College of Surgeons, for four days from Wednesday, April 16, through Saturday, April 19, at the John B. Murphy Memorial Auditorium, 40 East Erie Street, Chicago, Illinois.

SECTIONAL SURGICAL MEETING IN DES MOINES, IOWA

All members of the medical profession are invited to attend a three-day Sectional Meeting of the American College of Surgeons in Des Moines, Iowa, March 27 through 29, at the Hotel Fort Des Moines.

Topics will include emergency care of multiple injuries, surgery for congenital lesions, cardiac arrest, cancer, jaundiced patient, ovarian tumors, fluids and electrolytes. Medical motion pictures will also be shown daily, with an especially selected program scheduled for Thursday evening.

GENERAL MEDICINE AND SURGERY LECTURES

A three-day program of lectures and discussions on problems of current interest in general medicine and surgery will be presented by staff members of the Mayo Clinic and the Mayo Foundation for Medical Education and Research, April 14, 15 and 16, 1958. Up to twenty-one hours of Category I credit may be obtained by American Academy of General Practice members who attend. There are no fees for this program.

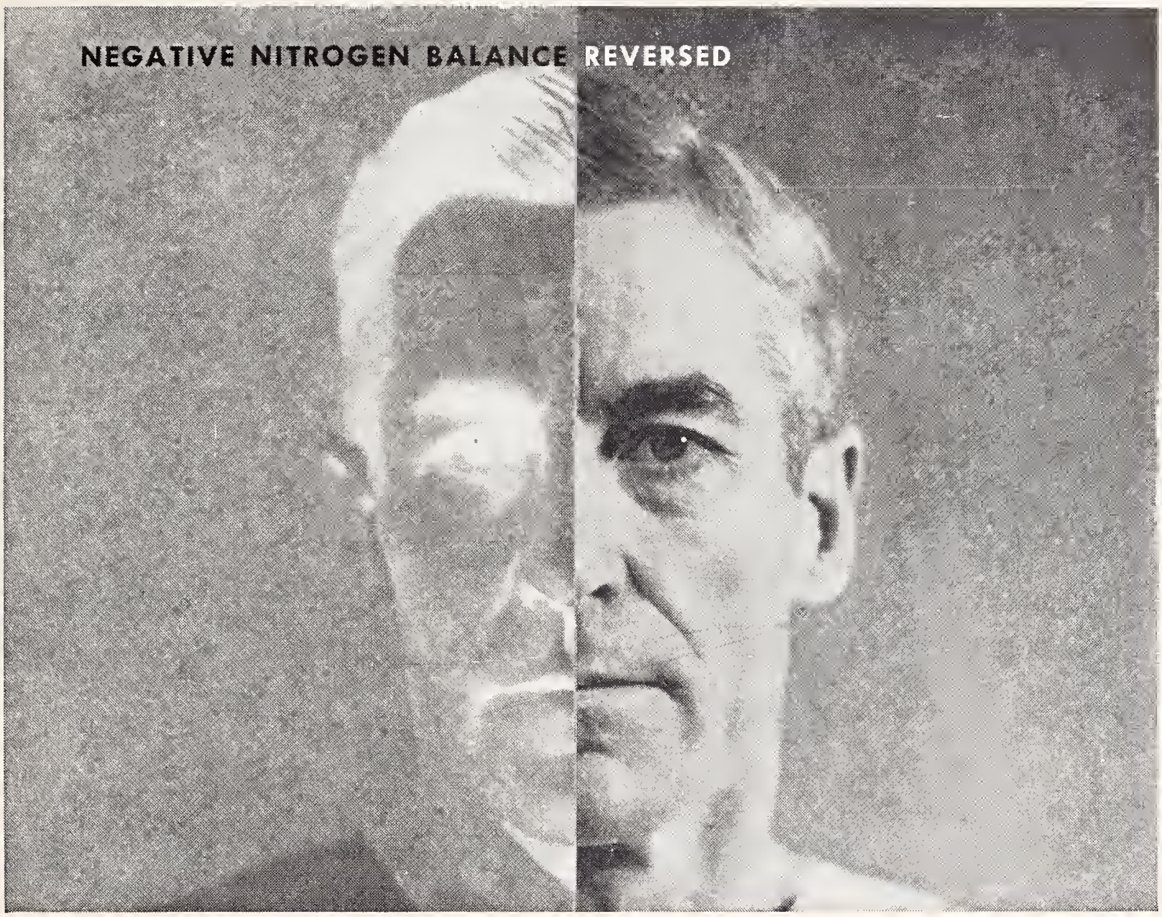
CONTINUATION COURSES

Medical continuation courses to be presented at the Center for Continuation Study, University of Minnesota, are as follows:

March 3-5	Pediatrics for General Physicians
March 17-19	Internal Medicine for Internists
March 20-22	Surgery for Surgeons
April 7-9	Radiology for General Physicians

For further information concerning the above courses, write to the Director, Department of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.

(Continued on Page A-42)



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stimulates protein synthesis,
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Increased nitrogen loss, with resulting negative nitrogen balance, occurs in infection, trauma, major surgery, extensive burns, certain endocrine disorders and starvation and emaciation syndromes. The intrinsic control of protein metabolism is lost and a protein "catabolic state" occurs. A patient requiring more than ten days of bedrest usually has had sufficient metabolic insult[1] to precipitate such a "catabolic" phase.

Nilevar (brand of norethandrolone) has been used in patients with varied conditions including hyperthyroidism, poliomyelitis, aplastic anemia, glomerulonephritis, anorexia nervosa and postoperative protein depletion. The patients gained weight and felt better. It was concluded[2] that "the drug certainly caused a reversal of rather recalcitrant or progressive catabolic patterns of disease."

Nilevar is unique among anabolic steroids in that androgenic side action is minimal or absent.

The suggested adult dosage is three to five tablets (30 to 50 mg.) daily. For children 1.5 mg. per kilogram of weight is recommended.

G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

---

[1] Axelrod, A. E.; Beaton, J. R.; Cannon, P. R., and others: Symposium on Protein Metabolism, New York, The National Vitamin Foundation, Incorporated, (March) 1954, p. 100.

[2] Proceedings of a Conference on the Clinical Use of Anabolic Agents, Chicago, Illinois, G. D. Searle & Co., April 9, 1956, pp. 32-35.

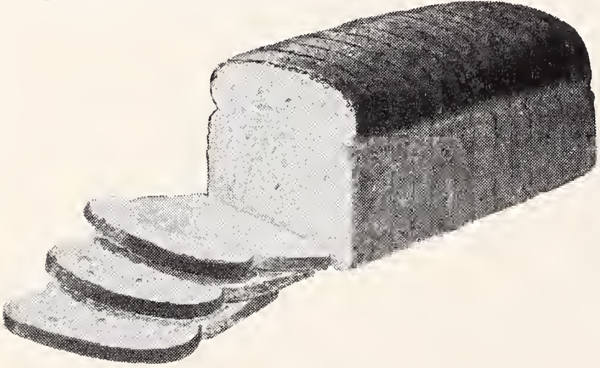
SEARLE

V^{10} protein
is a
COMPLETE PROTEIN

Complete protein is essential to the maintenance of body cells

Constant daily replacement of protein forming the cells of blood, skin, muscle, nerve, bone, and even teeth, is necessary to maintain health and vigor. In order that this process be maintained, the diet must contain adequate quantities of "complete protein" with all of the essential amino acids for simultaneous ingestion.

The Wisconsin Alumni Research Foundation has licensed the production of such a complete protein in the form of V^{10} Protein Concentrate. V^{10} Protein is composed entirely of grains, yet results of laboratory tests by the Foundation show that it has a protein efficiency value equal to casein, the high quality protein standard commonly used in protein evaluation work.*



Now V^{10} Protein is available in Minnesota in V^{10} Protein Bread. This light, delicious bread adds variety to the daily dietary requirement for protein. V^{10} Protein Bread will greatly aid in the planning of meals and will help promote health and vigor for all age groups.

WISCONSIN
ALUMNI
RESEARCH
FOUNDATION

*A complete report on these animal feeding studies is available on request. Address WISCONSIN ALUMNI RESEARCH FOUNDATION, P. O. Box 2217, Madison 1, Wis.

(Continued from Page 146)

THREE REGIONAL INSTITUTES ON PRINCIPLES OF EPIDEMIOLOGY

Three Regional Institutes on Principles of Epidemiology will be sponsored by the Minnesota Department of Health and University of Minnesota, School of Public Health, School of Veterinary Medicine in co-operation with the U. S. Department of Health, Education and Welfare, Public Health Service, Bureau of State Service, Communicable Disease Center Training Branch.

Dates and locations for the institute are as follows:

Fergus Falls	February 25, 26, 27, 28, 1958
Duluth	March 3, 4, 5, 6, 1958
Mankato	March 4, 5, 6, 7, 1958

The course is designed for health officers, physicians, nurses, veterinarians, laboratory workers, environmental health personnel and other members of the public health.

For additional information, write to D. S. Fleming, M.D., Director, Division of Disease Prevention and Control, Minnesota Department of Health, University Campus, Minneapolis 14, Minnesota.

COMMITTEE ACTION

Child Health Committee

(Continued from Page 139)

2. *Promotional*

   (a) Make surveys to ascertain what children are not now receiving continuous health supervision

   (b) Cooperate with other agencies in promoting the planned program of periodic health appraisal most suitable to any given community at the time of study

   (c) Assist in developing school health councils or subcouncils on school health under already existing community councils

   (d) Irrespective of what special medical examinations a child may need, recommend that primary general examination be made by the family physician or pediatrician

   (e) Aid in development of cumulative child health records and their use by parents, physicians, dentists, and schools

   (f) Promote research in child health programs as cooperative activities involving medicine, education, and public health

The Committee on Child Health of the Minnesota State Medical Association takes this opportunity to endorse these recommendations.

ELDON BERGLUND, M.D.
Liaison Representative to the PTA Committee on Child Health

when are tranquilizers indicated in pediatrics ?

Some doctors have questioned the use of tranquilizers in children. They feel, and rightly so, that these drugs should not be used as palliatives to mask distressing symptoms, while etiological factors go uncorrected. But there are three situations in which even the most conservative physician would not hesitate to use tranquilizers:

1. When the usually well-adjusted child needs a buffer against temporary emotional stress, such as hospitalization.
2. When a child needs relief from an anxiety-reaction that is in turn anxiety-provoking, so as to pave the way for basic therapy.
3. When anxiety underlies or complicates somatic disease, as in asthma.

In such situations, tranquilizers are likely to be more effective and better tolerated than previously accepted therapy, such as barbiturates.

But the question arises: **which** tranquilizer is suitable for children?

Most of the physicians now using tranquilizers in pediatric practice have found the answer to be ATARAX, confirming the conclusions of repeated clinical studies.

ATARAX is effective in a wide range of pediatric indications.
ATARAX has produced a "striking response" in a wide range of hyperemotive states.* In a study of 126 children, "the calming effect of hydroxyzine (ATARAX) was remarkable" in 90%.* Among the conditions that are improved with ATARAX are tics, nervous vomiting, stuttering, temper tantrums, disciplinary problems, crying spasms, nightmares, incontinence, hyperkinesia, etc.*

ATARAX is well tolerated even by children.
"ATARAX appears to be the safest of the mild tranquilizers. Troublesome side effects have not been reported. . . ."*

ATARAX offers two pediatric dosage forms.
ATARAX Syrup is especially designed for acceptability by medicine-shy youngsters. A small 10 mg. tablet is also available. In either case, you will get a rapid, uncomplicated response. Why not, for the next four weeks, prescribe ATARAX for **your** hyperemotive pediatric patients. See whether you, too, don't find it eminently suitable.

*Documentation on request

PEACE OF MIND ATARAX®
(BRAND OF HYDROXYZINE)

ATARAX
in any
hyperemotive
state

for childhood behavior disorders
10 mg. tablets—3-6 years, one tablet t.i.d.; over 6 years, two tablets t.i.d. Syrup—3-6 years, one tsp. t.i.d.; over 6 years, two tsp. t.i.d.

for adult tension and anxiety
25 mg. tablets—one tablet q.i.d. Syrup—one tbsp. q.i.d.

for severe emotional disturbances
100 mg. tablets—one tablet t.i.d.

for adult psychiatric and emotional emergencies
Parenteral Solution—25-50 mg. (1-2 cc.) intramuscularly, 3-4 times daily, at 4-hour intervals. Dosage for children under 12 not established.

Supplied: Tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

M. A. Seidell M.D.
Medical Director

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F. H. Magney, M.D., Secretary

Minneapolis Nurse Sentenced On Basic Science Charge

On December 2, 1957, a registered nurse, Mrs. Lois Kieser, twenty-six years of age, 5608 Osseo Road, Minneapolis, was sentenced by the Hon. Harold N. Rogers, Judge of the District Court of Hennepin County, to a term of one year in the Hennepin County jail. However, the Court then stayed the execution of the sentence for a period of one year and placed the defendant under the supervision of the Hennepin County Probation Office during this time. Judge Rogers imposed two conditions in connection with the stay of execution: (1) that the defendant refrain from the personal use of narcotic drugs except under the direction and prescription of a physician and, (2) that she consult a doctor of medicine once each month in reference to her health and authorize him to make a monthly report to the probation office. Mrs. Kieser was originally charged in this case with a felony, obtaining narcotic drugs by the forgery of a prescription. However, the charge was later reduced by the Court to practicing healing without having a certificate of registration in the basic sciences and Mrs. Kieser entered a plea of guilty before Judge Rogers to the reduced charge on September 20, 1957.

The defendant was arrested on September 17, 1957, after a Robbinsdale, Minnesota, pharmacist reported that he had delivered a 30 cc. vial of demerol to a woman at the Kieser home and received in exchange for the demerol a narcotic prescription purportedly signed by a Minneapolis doctor of medicine. When the physician was later contacted, he stated that the entire prescription was a forgery. A criminal complaint charging the defendant with the commission of a felony, as stated above, was then signed by a representative of the Minnesota State Board of Medical Examiners, the complaint being issued by the office of the Hennepin County attorney. When Mrs. Kieser was arrested she admitted forging not only the prescription in question but also five or six other prescriptions for demerol. In a signed statement, Mrs. Kieser said that she was born in Rock Elm Township, Wisconsin, on October 25, 1931, and is a registered nurse in the State of Minnesota. According to her statement, the defendant first began the personal use of demerol in February, 1957, and obtained some of the drug in a Minneapolis hospital where she was employed, by diverting to her own use a portion of the demerol that had been ordered for patients.

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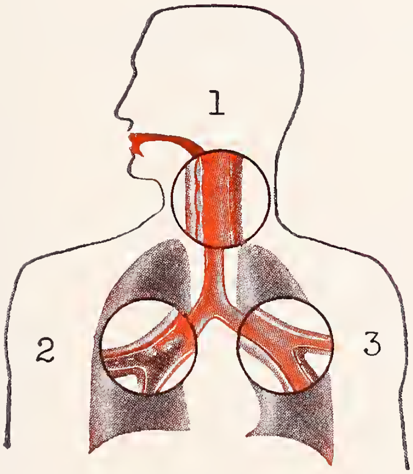
Radium element prepared in type of applicator requested

ORDER BY TELEPHONE OR MAIL
PRICES ON REQUEST

breaks up cough*



*Drawing shows how 3-pronged attack of Pyribenzamine Expectorant with Ephedrine breaks up cough by: (1) reducing histamine-induced congestion and irritation throughout the respiratory tract; (2) liquefying thick and tenacious mucus; (3) relaxing bronchioles. Pyribenzamine Expectorant with Codeine and Ephedrine also available (exempt narcotic). Pyribenzamine® citrate (tripelennamine citrate CIBA). **CIBA**

Woman's Auxiliary

ZUMBRO VALLEY AUXILIARY MEETS

The members of the Zumbro Valley Medical Auxiliary met at the Mayo Foundation House January 8 at 2:30 p.m.

Tea was served, followed by a business meeting.

Dr. Frank Krusen, head of the Mayo Clinic Section of Physical Medicine and Rehabilitation, spoke on the progress of the Ability Building Center in Rochester. Mr. David Griggs, Director of the Center, also reported on the activities of the past year.

RAMSEY COUNTY AUXILIARY NEWS

The Ramsey County Auxiliary resumed its activities after the holiday season with a board meeting on January 13 and a luncheon a week later in the Minnesota Club. The two tuberculosis essay winners, students at St. Agnes High School, attended and were presented with cash awards contributed by the Auxiliary and the Ramsey County Tuberculosis and Health Association. Three students from the American Field Services were introduced by Mrs. M. M. Sarnecki, who is affiliated with the service in St. Paul. Two were natives of the Philippines and Iceland, and the third, David Nelson, Minneapolis, of St. Thomas College, spent the summer in Denmark. These young people gave interesting accounts of their experiences. The Mmes. H. O. Peterson, Herman Wolff, and O. I. Sohlberg, the committee in charge of the AAPS essay contest, reported that they are putting notices in the high schools to stimulate interest in the contest.

Mrs. C. L. Oppegaard, state president, was honor guest at the luncheon. The president greeted the members, urging everyone to attend the Mid-Winter Board meeting in the Minnesota Club. Tribute was paid to the memory of a former president, Mrs. A. G. Schulze, who died recently. Mrs. Schulze served very ably from 1927-29. The Auxiliary accepted the board's recommendation to send a bequest in memory of Mrs. Harold Wahlquist, to the State Auxiliary to be used as the state committee sees fit. It was also decided to give a grant of $100 to KTCA for the purpose of furthering educational TV programs.

SEASON'S GREETINGS FROM FORMOSA

"The red color of the card upon which this greeting is written is the favorite color for festivals, and firecrackers are in vogue for every occasion. Our New Year is celebrated but their 'New Year' is really celebrated. It will begin February 18 and then it's a continuous feasting and everyone goes in debt again till the next year's end, when checks are issued to pay the bills (many rubber ones)!! So it's a continuous 'being in the red!!!'

"It seems there is a story with every patient—each one knows that he cannot pay for the medicine used, so when a live chicken is brought to the door we know, too, that the family is offering a real sacrifice in appreciation for health.

"We think of all of our dear friends very often and we wish you all the joys of the season."

THEO and P. J. PANKRATZ

(Text of a greeting to the auxiliary from Mrs. Pankratz to Mrs. A. B. Rosenfield.)

MARGARET SHEMAN WAHLQUIST MEMORIAL COMMITTEE NAMED

A committee to consider an appropriate memorial in tribute to Mrs. Harold Wahlquist, state and national auxiliary leader, who died August 9, 1957, has been named by Mrs. C. L. Oppegaard, state auxiliary president.

Memorial possibilities suggested to date include contributions to cancer research or to the American Medical Education Foundation Fund. A memorial trust fund for scholarships bearing the name of Mrs. Wahlquist has also been suggested. Contributions will be received on a voluntary basis.

Members of the committee are: Mrs. L. P. Howell, Rochester; Mrs. H. H. Fesler, St. Paul; Mrs. Harold Benjamin, Minneapolis; Mrs. Peter Rudie, Duluth; and Mrs. O. M. Heiberg, Worthington.

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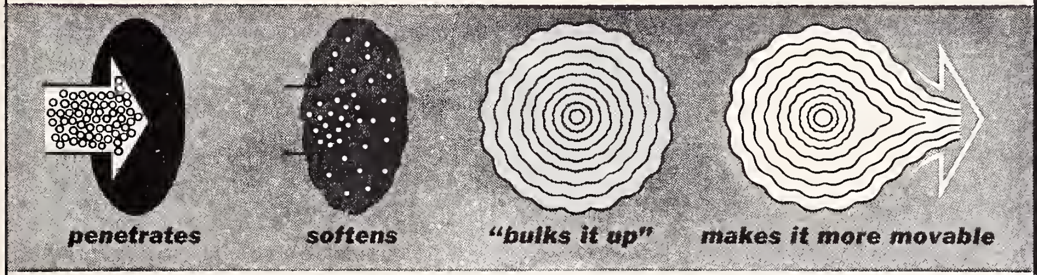
permeates the hard, stubborn stool of chronic constipation with millions of microscopic oil droplets, each encased in a film of Irish moss... makes it more movable



KONDREMUL *(Plain)*—Pleasant-tasting and non-habit-forming. Contains 55% mineral oil. Supplied in bottles of 1 pt.

KONDREMUL *(With Cascara)*—0.66 Gm. nonbitter Ext. Cascara per tablespoon. Bottles of 14 fl.oz.

KONDREMUL *(With Phenolphthalein)*—0.13 Gm. phenolphthalein (2.2 gr.) per tablespoon. Bottles of 1 pt.

When taken as directed before retiring, KONDREMUL does not interfere with absorption of essential nutrients.

THE E. L. PATCH CO. — STONEHAM, MASSACHUSETTS

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In Memoriam

MARTIN AUNE

Dr. Martin Aune, eighty-four, died January 4, 1958. A practicing Minneapolis physician for fifty years, Dr. Aune was a member of Hennepin County Medical Society, Theta Kappa Psi medical fraternity, Minneapolis Lions Club, Minnesota Chapter of National Sojourners, Khurum Masonic Lodge 112, Zuhrah Temple and Scottish. He was also a life member of the Minnesota State Medical Association.

Dr. Aune, lieutenant colonel on the general staff of the Minnesota National Guard, and a World War I veteran, was appointed medical director of the Twin City Ordinance Plant Hospital in New Brighton in 1941. He retired from National Guard service in 1944 and was appointed as state surgeon of the Minnesota Defense Force with rank of lieutenant colonel in 1946.

Survivors include his wife, Leah; four sisters, Mrs. Lucy Adams, Emma and Alma Aune, Duluth, Minn., and Mrs. Arthur Asplund, Milwaukee, Wisconsin.

HAROLD L. BOLENDER

Dr. Harold L. Bolender, fifty-five, prominent St. Paul physician, died January 6, 1958 at Midway Hospital in St. Paul where he had been hospitalized for nearly three weeks.

He had practiced medicine in St. Paul since his graduation from the University of Iowa in 1930. His special work included one year of internship at Ancker Hospital in St. Paul.

A native of Northwood, Iowa, he received his preliminary education in the public schools of that community.

Dr. Bolender was a member of the Ramsey County Medical Association, an active member of the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife, Hazel; two daughters, Jean and Sandra, both of St. Paul; his mother, Mrs. Lizzie Bolender of Northwood, Iowa; and two brothers.

GEORGE R. FUGINA

Dr. George R. Fugina, a physician and surgeon in Mankato and Madison Lake for nearly fifty years, died January 10, 1958. He was seventy-two years old. He served on the staff of St. Joseph's Hospital since 1909 and was on the Immanuel Hospital staff since 1920.

Dr. Fugina had been practicing medicine in Mankato since 1924 when he moved from Madison Lake. He practiced in Madison Lake since 1909.

He was born in Fountain City, Wisconsin. He was graduated from the College of Physicians and Surgeons at the University of Illinois in 1908. He served his internship at St. Joseph's Hospital, Ashland, Wisconsin.

He was a life member of the Blue Earth County Medical Society, the Minnesota State Medical Association, the American Medical Association and the Catholic Order of Foresters.

His survivors include his wife, Maria; one daughter, Mrs. Lawrence A. Atwell, Arlington, Virginia; one sister, Miss Laura Fugina, Fountain City; and two great-grandchildren.

ANDREW W. HILGER

Dr. Andrew W. Hilger, seventy-eight, pioneer St. Paul ear, nose and throat specialist, died January 6, 1958.

A native of Bismarck, North Dakota, Dr. Hilger had practiced medicine in St. Paul for fifty years. He was a graduate of the University of Minnesota. Dr. Hilger was a member of the Ramsey County Medical Association and a life member of the Minnesota State Medical Association.

Surviving are his wife, Winnifred; two sons, Andrew W. Hilger, Jr., and Dr. John R. Hilger, both of St. Paul; a daughter, Mrs. Harold Thibault, Coronado, California; two brothers, Robert and John, and a sister, Mrs. Robert Prendergast, all of St. Paul; and two grandchildren.

ALVAH W. JONES

Dr. Alvah W. Jones, pioneer Red Wing physician, died January 4, 1958, at the age of ninety-four.

Dr. Jones was born in Fulton County, Ohio. He attended high school in Wauseon, Ohio, and came to Red Wing in 1882. He studied at the University of Minnesota for three years.

During the years of 1885 and 1886 Dr. Jones taught at Tower School and later became an instructor at the Hauge Seminary in Red Wing. In the fall of 1887 Dr. Jones enrolled at Rush Medical College and later that year accepted a position as special pension examiner for the United States government.

In 1888 he entered Georgetown University and was graduated as a doctor of medicine in 1891. He was valedictorian of his graduating class.

During his many years of practice in Red Wing, Dr. Jones served as city health officer for twenty-one years and for fifteen years was a member of the Red Wing board of education. He was also active in the Masonic Lodge, being a member of Red Wing Lodge, No. 8, A.F. and A.M.

Dr. Jones was a member of the Goodhue County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife and one son, Frederick W. Jones of San Leandro, California.

(Continued on Page A-50)

CLINICAL COLLOQUY

*My patients complain that
the pain tablets I prescribe
are too slow-acting...
they usually take about
30 to 40 minutes to work.*

**Why don't you try
the new analgesic
that gives faster,
longer-lasting pain relief?**

*What is it...
how fast does it act?*

**It's Percodan®—relieves pain
in *5 to 15 minutes*,
with a single dose
lasting 6 hours or longer.**

How about side effects?

**No problem. For example,
the incidence of constipation
with Percodan* is rare.**

*Sounds worth trying—
what's the average adult dose?*

**One tablet every 6 hours.
That's all.**

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WILLIAM WINFRED KLIMA

Dr. William Winfred Klima, seventy-three, of Stewart, died December 11, 1957, in the Hutchinson Community Hospital where he had been a patient since July.

At the time of his retirement in August, 1956, he had been a practicing physician in the Stewart community for thirty-seven years.

A native of Silver Lake, he attended Hutchinson public schools. Prior to attending the University of Minnesota, Dr. Klima taught in rural schools in the Hitchinson area for several years. He was graduated from the University of Minnesota in 1917.

For one year after his graduation Dr. Klima interned at Ancker Hospital in St. Paul. He practiced for one year in his home community of Silver Lake. He opened his practice in Stewart in 1919.

Dr. Klima was active in the civic life of Stewart. He was a former member of the Stewart City Council. During World War II he was actively engaged in conducting community first aid courses.

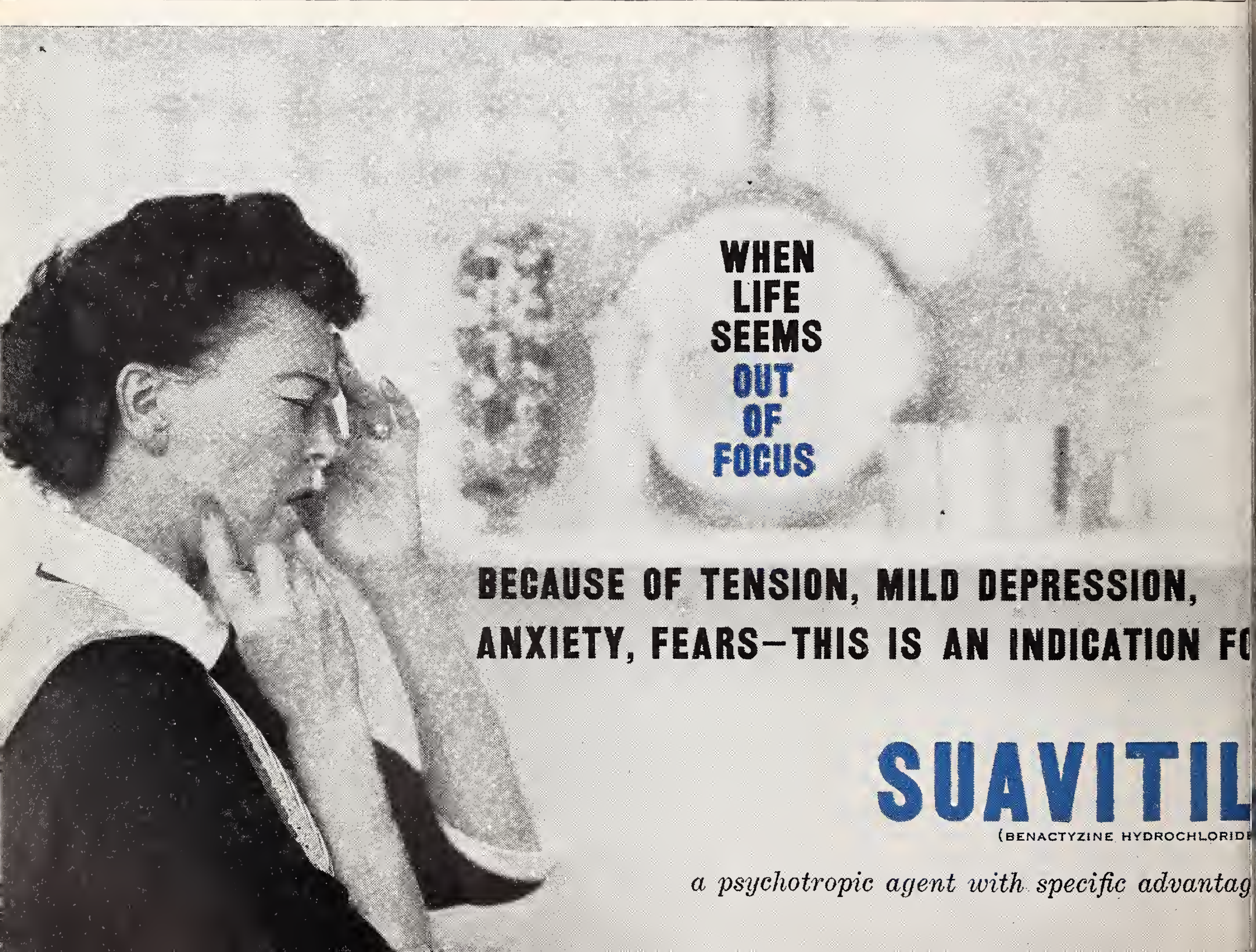
He was a member of the McLeod County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife, Martha; two daughters, Mrs. Tom Koeppen, Hutchinson and Mrs. Howard Morton, Moline, Illinois; seven grandchildren; and one sister, Mrs. Frances Truhlar of St. Paul.

EUGENE T. LEDDY

Dr. Eugene Thomas Leddy, widely known specialist in x-ray treatment and a member of the staff of the Mayo Clinic for thirty-two years, died at St. Mary's Hospital in Rochester, Minnesota, on January 5, 1958. He had retired from active practice on October 1, 1957. Dr. Leddy was born in Taunton, Massachusetts. He received the degree of bachelor of arts in 1915 and that of doctor of medicine in 1919, both from Harvard University. He was an intern at Memorial Hospital in New York City for three months, and at Carney Hospital in Boston for 6 months. From 1920 to 1922 he was an assistant at the Memorial Hospital, and from 1922 to 1925 he was head of the roentgen-ray department of the Philadelphia General Hospital. For six months in 1925, he carried on the private practice of roentgenology in Providence, Rhode Island, where he was head of the x-ray department at the Rhode Island Hospital.

Dr. Leddy came to Rochester, Minnesota, on October 1, 1925, as a first assistant in therapeutic roentgenology. On April 1, 1929, he was appointed to the staff of the Mayo Clinic as a consultant in therapeutic roentgenology and as an instructor in radiology in the Mayo Foundation, Graduate School, University of Minnesota. He was advanced to assistant professor in 1933 and associate professor in 1937. He was certified as a specialist in radiology in 1934 by the American Board of Radiology, Inc. He was appointed head of the Section of Thera-


WHEN
LIFE
SEEMS
OUT
OF
FOCUS
BECAUSE OF TENSION, MILD DEPRESSION,
ANXIETY, FEARS—THIS IS AN INDICATION F
SUAVITIL
(BENACTYZINE HYDROCHLORIDE
a psychotropic agent with specific advantag

peutic Radiology at the Mayo Clinic on July 1, 1948, a post he held until April 1, 1953, when he became a senior consultant in therapeutic radiology.

Dr. Leddy, almost from the time of his graduation from medical school, devoted his career to the treatment of various conditions, especially malignancies, with x-rays and radium. He was regarded as a pioneer in the medical uses of radium.

Dr. Leddy was a fellow of the American College of Radiology and a member of the Zumbro Valley Medical Society, an associate member of the Minnesota State Medical Association, the American Medical Association, the American Roentgen Ray Society, the Radiological Society of North America, the American Radium Society, the Alumni Association of the Mayo Foundation and the Society of the Sigma Xi. He was an honorary member of the Old Blockley Medical Society of Philadelphia.

Dr. Leddy was married to Miss Anna C. Regan on June 8, 1926. Mrs. Leddy and two children survive him: John Eugene Leddy and Anna Marie Leddy.

PETER MILTON MATTILL

Dr. Peter Milton Mattill, seventy, assistant superintendent and associate medical director of Glen Lake sanatorium since 1938, died January 12, 1958 in Northwestern Hospital in Minneapolis.

Dr. Mattill, a resident at the sanatorium, had served on its staff since 1924.

Born in O'Daniel, Texas, he received his preliminary education at Albany, Missouri, and St. Joseph, Missouri, high schools. His bachelor of science degree was conferred at North Central College, Naperville, Illinois, and his master of science degree at the University of Chicago. He received his medical education at Rush Medical College in Chicago.

Dr. Mattill was a staff member of Children's Memorial Hospital, Chicago, and Rood Hospital, Hibbing-Chisholm, Minn., before joining the Glen Lake institution as a staff physician.

The American College of Physicians made him a fellow in 1929. He also belonged to American Board of Internal Medicine and American Trudeau Society. He was president of the Minnesota chapter in 1952-53.

Dr. Mattill was clinical assistant in medicine at University of Minnesota School of Medicine from 1926 to 1946 and instructor in the University's School of Nursing from 1946 to 1956.

His memberships include the Hennepin County Medical Society, Minnesota State Medical Association, American Medical Association, Minneapolis Society of Internal Medicine, Gethsemane Lutheran Church and Glen Lake School Board.

Surviving Dr. Mattill are his wife, Nora A.; four daughters, Mrs. John R. Holum, Minneapolis, Mrs. Herbert Strom, Red Lodge, Montana, Barbara and Phyllis Mattill, Oak Terrace; three brothers, Andrew, St. Joseph, Missouri, Charles, Missoula, Montana, and Henry, San Diego, California; and a sister, Emma Mattill, Upland, California.

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ntly, gradually, without euphoric buffering,
VITIL helps patients recover normal drive and
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OMMENDED DOSAGE: 1.0 mg. t.i.d. for two or three
s. If necessary this dosage may be gradually
eased to 3 mg. t.i.d.

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General Interest

Dr. R. B. Skogerboe has announced that **Dr. Bruce Boynton** of Grafton, North Dakota, has joined the staff at the Karlstad Memorial Hospital.

* * *

Dr. Kermit Halvorsen joined the Mesaba Clinic in Chisholm the first of January.

* * *

Three Mayo Clinic doctors participated in programs at two sectional meetings of the American College of Surgeons in January. **Dr. James H. DeWeerd** collaborated in a panel discussion on automotive injuries at a meeting at Dallas, Texas. **Dr. Malcolm B. Dockerty** participated in a symposium on "Studies on Control of Spread of Cancer" at the same meeting. **Dr. John M. Waugh** of the Mayo Clinic participated in the three-day sectional meeting of the College at Jackson, Mississippi.

* * *

Dr. B. A. Gingold, Minneapolis, has been elected president of Asbury Methodist Hospital medical staff for 1958. **Dr. C. V. Kusz** is vice president and **Dr. S. R. Maxeiner, Jr.**, secretary-treasurer. Others named to the executive committee are **Dr. C. A. Neumeister**, chief of surgery; **Dr. D. R. Hastings**, chief of medicine; **Dr. M. E. Baker**, chief of obstetrics and gynecology, and **Dr. William H. Rock**, chief of general practice.

* * *

Dr. Frank H. Krusen, head of the Section of Physical Medicine and Rehabilitation at the Mayo Clinic, has been reappointed by Gov. Orville Freeman to the State Board of Health for a term ending January 1, 1961. This is Dr. Krusen's second term.

DELINQUENT

"ABOUT THIS JONES ACCOUNT, DOCTOR. WHAT ARE THE CHANCES FOR RECOVERY?"

* * *

Dr. C. W. Truesdale has sold his interest in the Glencoe Clinic to **Dr. M. M. Howell** effective January 1. However, Dr. Truesdale will continue to hold office hours at the Glencoe Clinic until his office quarters are opened in Glenhaven which will probably be some time in February.

* * *

Dr. Richard M. Hewitt, head of the Mayo Clinic Section of Publications for fifteen years, has been appointed the third Alfred P. Sloan Visiting Professor at the Menninger Foundation at Topeka, Kansas.

* * *

Dr. Harold E. Miller was re-elected president of the medical staff of Northwestern Hospital. Others renamed to the staff are **Dr. Albert J. Schroeder**, secretary-treasurer; **Dr. Mark C. L. Hanson**, chief of medicine, and **Dr. Edgar Ingalls**, chief of obstetrics and gynecology. New officers are **Dr. Maynard C. Nelson**, vice president; **Dr. Harold Buchstein**, chief of surgery; **Dr. Northrop Beach**, chief of pediatrics, and **Dr. Frederick Hass**, chief of general practice.

* * *

The new offices of **Drs. M. A. and F. M. Burns** are located on Main Street in Milan. Formerly the Milan Hotel, the building was purchased some time ago by **Dr. F. M. Burns** and the first floor was remodeled for the doctors' offices.

* * *

Dr. John S. Lundy, senior consultant in anesthesiology in the Mayo Clinic, Rochester, and professor of anesthesiology in the Mayo Foundation, Graduate School, University of Minnesota, was the recipient of the second Award of Merit of the Horace Wells Club of Connecticut at the sixty-third annual meeting of the organization of the club in Hartford. The award was given to Dr. Lundy for his outstanding contributions to anesthesiology during his career.

* * *

Dr. J. S. Blumenthal, Clinical Associate Professor, Department of Medicine and Chief of Allergy Clinic, University of Minnesota Hospitals, has been appointed a member of the National Committee on Allergy of the American College of Chest Physicians.

* * *

Dr. Edward A. Pasek, Faribault, has recently been certified by the American Board of Ophthalmology.

* * *

Dr. Gordon Erskine, Grand Rapids physician, has announced his retirement after practicing in that community for almost twenty years. He is a former chief of staff at Itasca Memorial Hospital.

(Continued on Page A-54)

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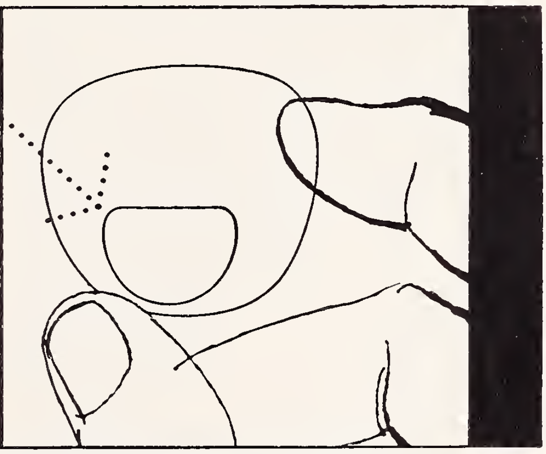

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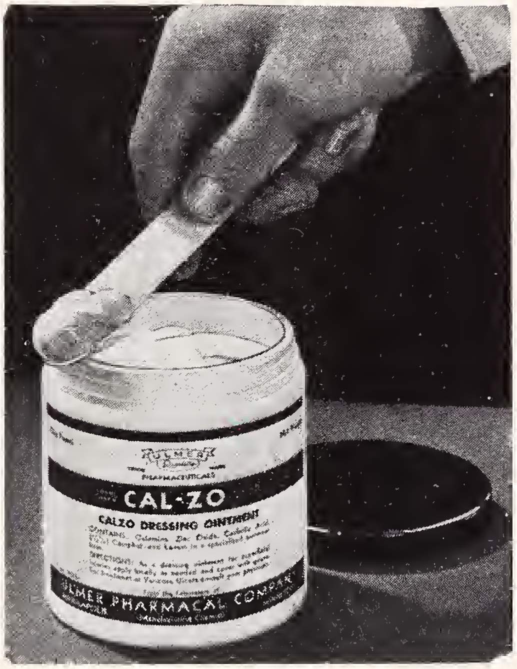

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(Continued from Page A-52)

Dr. Barnard Hall has been elected chief of staff at St. John's Hospital in St. Paul. **Dr. Paul Polski** was elected first vice chief of staff and **Dr. Paul Rick** was elected second vice chief. **Dr. M. G. Bernier** was named secretary. Elected to the executive committee were **Dr. A. E. Muller** and **Dr. C. R. Tift.** Dr. Hall announced appointment of **Dr. C. K. Cook** as chief of surgery, **Dr. M. E. Janssen,** chief of medicine; **Dr. Jack Strand,** chief of physical medicine and rehabilitation; **Dr. R. W. Emmons,** chief of obstetrics, and **Dr. Herbert Stolpestad,** chief of general practice.

* * *

Dr. John Pewters, president of the medical staff of Abbott Hospital, St. Paul, and the entire hospital executive board were re-elected for another year. **Dr. S. L. Arey** is vice president and chairman of the pediatrics division; **Dr. John Johnson,** secretary and chairman of medicine. Executive board members are **Dr. Tague C. Chisholm,** head of surgery; **Dr. Karl E. Sandt,** head of eye, ear, nose and throat division, and **Dr. Richard R. Fliehr,** in charge of obstetrics and gynecology.

* * *

"Medical Men of Meeker County" is the title of a book compiled by **Dr. H. E. Wilmot** of Litchfield.

* * *

Dr. Arthur Larson and his wife, **Dr. Dorette Larson,** Madison, have left the staff of the Fergus Falls State Hospital after serving there for eight months. **Dr. Arthur Larson** will spend three years at the University of Minnesota under a fellowship in pathology.

* * *

Dr. David E. Ellison, Minneapolis, has been named the president of the Twin City Rapid Transit Company.

* * *

Dr. R. B. Pierce, Renville physician, has been granted a patent for a device to hold fractured bones in place. The device was developed after long study and research and incorporates the use of stainless steel pins to anchor the bones at the fracture.

* * *

New officers of the West Central Minnesota Medical Society include **Dr. Irwin Oliver,** president, and **Dr. Carl Swendseen,** secretary-treasurer. Both doctors practice in Graceville.

* * *

The University of Minnesota has received more than three million dollars in medical research grants during the twelve months ending June 30, 1957.

The federal public health service granted $1,625,000 of the $3,060,000 total. Other grants brought the total federal government participation to $1,902,000.

Private foundations outside Minnesota contributed $606,482; and private sources in Minnesota contributed $469,388. The state legislature appropriated $82,500 for medical research.

* * *

Dr. Robert R. Kierland, consultant in the section of dermatology, Mayo Clinic, Rochester, has been elected secretary-treasurer of the American Academy of Dermatology and Syphilology.

Dr. Richard Bardon has been named president-elect of the St. Louis County Medical Society for 1959. **Dr. J. C. Feuling** was installed as president for 1958. He succeeds **Dr. Earl E. Barrett.** Other officers named were **Drs. W. H. Parker,** Chisholm, vice president; and **R. O. Bergan,** secretary-treasurer.

* * *

Dr. F. A. Meyerding will retire as executive secretary of the Minnesota Tuberculosis and Health Association April 1. His successor will be Mr. J. G. Neal, supervisor of health, physical safety and recreational education for the state of Minnesota.

* * *

Rochester's newly appointed civil defense director is **Dr. Winchell McK. Craig,** a retired Mayo Clinic neurosurgeon and retired rear admiral in the Naval Medical Corps Reserve.

* * *

A medical-surgical building will be named for **Dr. George E. Fahr,** head of medicine at Minneapolis General Hospital for twenty-four years, staff member since at the Anoka Hospital. Dr. Fahr was recently honored for his contribution in the development of the electrocardiogram.

* * *

The McLeod County Medical Society has elected **Dr. M. M. Howell,** president and **Dr. C. W. Truesdale,** secretary-treasurer. Both doctors are residents of Glencoe.

* * *

New president of the Winona County Medical Society is **Dr. Carl R. Heise.** He succeeds **Dr. Sidney O. Hughes.** Also elected were vice president, **Dr. L. J. Wilson,** succeeding **Dr. R. B. Tweedy;** secretary, **Dr. C. M. Johnson,** succeeding **Dr. Wilson,** and treasurer, **Dr. C. W. Rogers,** replacing **Dr. W. W. Haesly. Dr. L. I. Younger** was re-elected delegate to the Minnesota Medical Association and **Dr. H. W. Satterlee,** Lewiston, was re-elected.

* * *

Dr. Norbert O'Keefe, recently named chief of staff of the Olmsted County Community Hospital, will continue his practice in Spring Valley as well as being chief of staff of the hospital during 1958.

* * *

Mayo Clinic doctors who have recently retired include **Dr. Virgil S. Counseller,** head of the section of general surgery since 1928, and **Dr. Samuel F. Haines,** member of the clinic board of governors.

* * *

Dr. L. M. Eaton, head of the sections of neurology of the Mayo Clinic, has been elected president of the Association for Research in Nervous and Mental Diseases held recently in New York.

* * *

Dr. A. M. Lundholm, former St. Paul surgeon, is now associated with the Cambridge Clinic.

* * *

Dr. Richard A. DeWall, a Minnesota fellow in surgery at the University of Minnesota Hospitals, has been named one of the ten outstanding men of the year by the United States Junior Chamber of Commerce.

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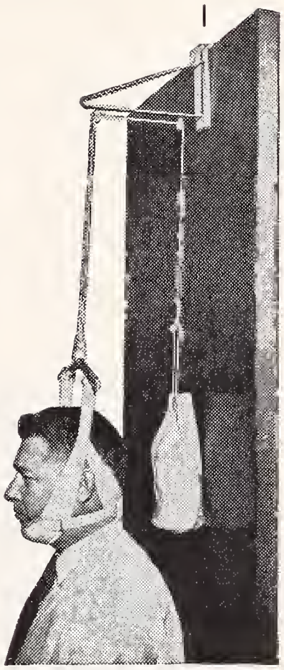

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Dr. DeWall distinguished himself for the discovery of a life-saving artificial heart-lung machine—called "bubble oxygenator." The device has been used in 357 open heart operations at University of Minnesota Hospital in the past two and one-half years.

* * *

Dr. C. A. Williams, Pipestone, has retired after devoting forty-five and one-half years to the medical profession. He has sold his practice to **Dr. R. J. Kotval**, who had been his partner since July 1, 1953.

* * *

Latest addition to the Olmsted Community Hospital staff in Rochester is **Dr. C. J. Corrigan**, a radiologist, formerly of Minneapolis and Saint Paul.

* * *

Dr. Ralph K. Ghormley of the Mayo Clinic staff recently participated in a discussion this month at the 18th annual Congress on Industrial Health in Milwaukee.

* * *

Dr. J. F. Haas, who previously was associated with **Dr. Glenn Nelson** in Fairfax, is now practicing with **Drs. S. T. Kucera** and **G. N. Rysgaard** in Northfield.

* * *

Dr. Harold S. Diehl has left University Hospitals at Minneapolis following his recovery from minor injuries suffered in an automobile accident the day before Christmas. Dr. Diehl, dean of the College of Medical Sciences at the University, suffered a dizzy spell at the wheel of his car and hit a bridge retaining wall. The car was badly damaged when it was wedged between the wall and a telephone pole. He suffered only minor injuries in the accident, but entered the hospital for a further checkup of his general condition. Recently, Dr. Diehl accepted the post as senior vice president for research and medical affairs and deputy executive vice president of the American Cancer Society. He has since taken over his new duties in New York.

AMA PRESIDENT-ELECT MAKES TWIN CITY APPEARANCES

"Medical Horizons" was the title of a convocation address presented by Dr. Gunnar Gundersen, president-elect of the American Medical Association at the fifty-sixth annual Farm and Home Week, held January 14-17 on the University of Minnesota St. Paul campus.

Minnesota's medical pioneers knew that good medical practice depends primarily on sound education, Dr. Gundersen told the convocation audience as he traced the various steps in the development of medical practice in Minnesota throughout the past century.

He also paid tribute to the Mayo Clinic at Rochester, Minnesota, saying the world-famed medical center grew out of the great character, human kindliness and profound sympathy of its founding brothers, Doctors Charles and William Mayo.

American society is discouraging bright young men from going into medicine, Dr. Gundersen told a press

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conference held in conjunction with his Twin City appearances. He emphasized unless something is done about it, America might find itself in the same situation as the Soviet Union, where eighty per cent of medical students today are women. The A.M.A. president-elect pointed out that immediately following World War II, there were five applicants for each opening in a United States medical school.

Today, he stated, there are less than two. However, he said, the situation after the war was unusual because there was a great backlog of young men whose college education had been delayed because of service. One of the things that make a medical center less attractive, is the growing encroachment of government, which is repugnant to independent, free-thinking Americans. One example he cited was government directed medical care of veterans. Medical care for service-connected disabilities properly is the concern of government, he said. But veterans should go to private practitioners for care of non-service-connected disabilities, he added.

He also cited the length of medical education—ten years—and its cost as factors which make young men hesitate about going into medicine. A medical education today costs $15,000, he said. Many young people feel they cannot afford such an investment, he continued, especially when they consider that the average productive life of a physician is twenty-five years.

Dr. Gundersen's recent Twin City appearance marks the first of several which he is expected to make in Minnesota during 1958. On June 14, he will present the address at the University of Minnesota Medical School's commencement exercises. During December, he will attend the American Medical Association clinical meetings which will be held in Minneapolis.

CORRECTION

In the Summary and Conclusion of the article entitled "A Practical Approach to the Suppression of Lactation," published in the November, 1957 number, pages 794-796, the dosage should read "*one* tablet three times a day for the first four days and one tablet daily for the ten days thereafter."

U.S. EXPECTS PHYSICIAN-DENTIST SUITS

(Continued from Page 137)

"It must have been an oversight," said the Court, "for it seems to us that any reasonable creature in being whose mind was not in a state of dover . . . would inevitably reach the conclusion that if plaintiff's services were to be continued after passage of the act of June 29, 1953, a commission would be required if the provisions of the law were complied with."

After June 29, 1953, the doctor-draft law required that inductees receive rank commensurate with experience and training. To date, the Army alone has uncovered eighteen potential plaintiffs.

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MINNESOTA BLUE CROSS-BLUE SHIELD

The Professional Relations Staff of Minnesota Blue Shield traveled over 25,000 miles during 1957 to visit doctors' offices throughout Minnesota and to inform physicians on Blue Shield matters. More visits to physicians' offices during 1957 resulted in personal visits with doctors than in any previous year since the beginning of the Professional Relations Program in 1953.

While claim matters are most frequent inquiries of physicians involves the proper handling of medical service reports for patients who are subscribers with other Blue Shield Plans. Such services can be reported on Minnesota Blue Shield Medical Service Reports, which include the information required by other Blue Shield Plans. Reports should then be mailed directly to the Blue Shield Plans in which the patients are subscribers. If the address of any Plan is not available, or if any other information is needed, Minnesota Blue Shield will gladly provide assistance.

During 1957, many physicians inquired about the availability of the Blue Shield coverage for themselves and their families. In February of 1956 the Blue Shield Board of Directors made Blue Shield contracts available to doctors, because it was found that many doctors considered Blue Shield coverage a method of solving some of the embarrassments arising from the courtesy care of one physician by another. A doctor who wishes to obtain a Blue Shield contract can do so by writing to Blue Shield, 2610 University Avenue, St. Paul 14, Minnesota.

Another matter about which physicians ask is Blue Shield enrollment on a national level or wherever Blue Shield Plans are in operation. As of September 30, 1957, a total of 73 Blue Shield Plans were in operation in 43 states, the District of Columbia, Puerto Rico, Hawaii, and Canada. Total enrollment as of this date was 40,686,426.

Hospital admission figures recently compiled by Minnesota Blue Cross indicate that upper respiratory infections; pneumonia, flu and the common cold hit Minnesota citizens harder during the months of October and November, than at any other time in the past few years.

Nearly one-fourth of the 26,203 respiratory cases incurring Blue Cross claims through the first eleven months of 1956 occurred during the months of October and November. During October and November, a total of 6,029 Blue Cross members were hospitalized for upper respiratory infections.

The obvious upswing in respiratory infections proved to be costly. Blue Cross hospitalization expense for respiratory illnesses of $260,000 and $390,000 during the months of October and November, respectively, exceeded the average cost per month of approximately $200,000 experienced through the first nine months of 1957.

Over-all Blue Cross hospitalization expense through the first eleven months of 1957, totaled $25,969,419, providing 1,122,141.6 days of hospital care for 185,449 participant subscribers.

Accidental injuries incurred by Blue Cross subscribers still remain the principle cause of hospitalization with 31,434 cases representing seventeen per cent of total cases paid to date.

Pregnancies and conditions relating to pregnancy involved 29,681 subscribers and represented sixteen per cent of all cases paid during the first eleven months of the year.

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

The Nineteenth Annual Meeting of the American Industrial Hygiene Association will be held in Atlantic City, New Jersey, April 21-26, 1958. More than one hundred technical papers will be presented on topics from the fields of radiation, air pollution, noise, toxicology, engineering control of hazards, and analytical chemistry. Professional hygienists and interested persons from industry, public health, labor and management groups, schools and universities, safety and technical organizations, medical institutions, and from the armed forces will convene to discuss current problems and recent advances in these fields.

Headquarters for the AIHA activities will be the Claridge Hotel. Registration will commence Sunday, April 20, at the Claridge Hotel and at Haddon Hall. Beginning Monday, April 26, registration will continue at the Convention Hall.

Advance registration may be obtained by mailing $3.00 to the Industrial Health Conference Registration, Room 1300, 28 E. Jackson Blvd., Chicago 4, Illinois.

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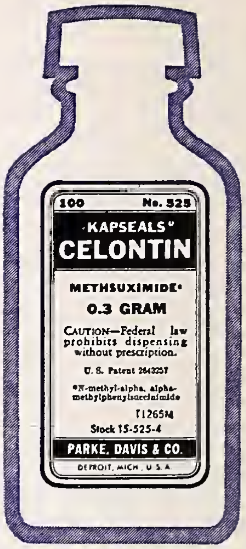
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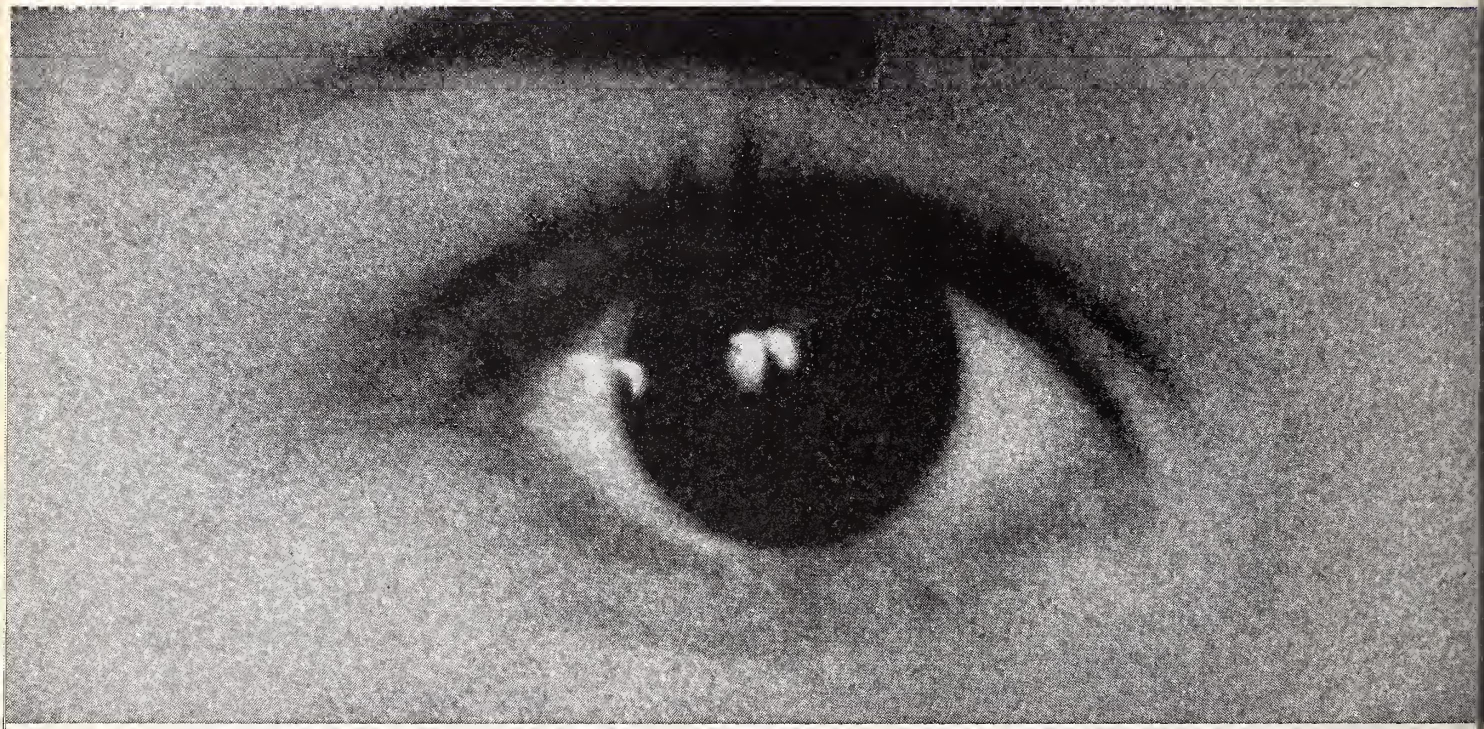

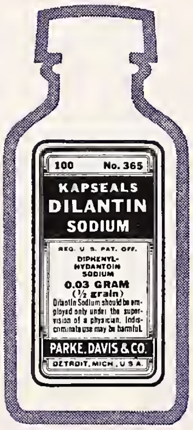
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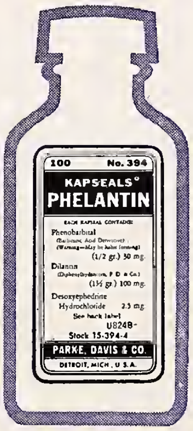
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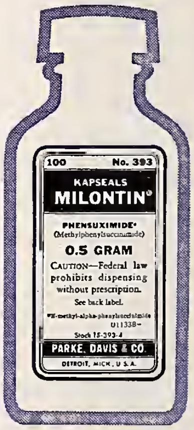
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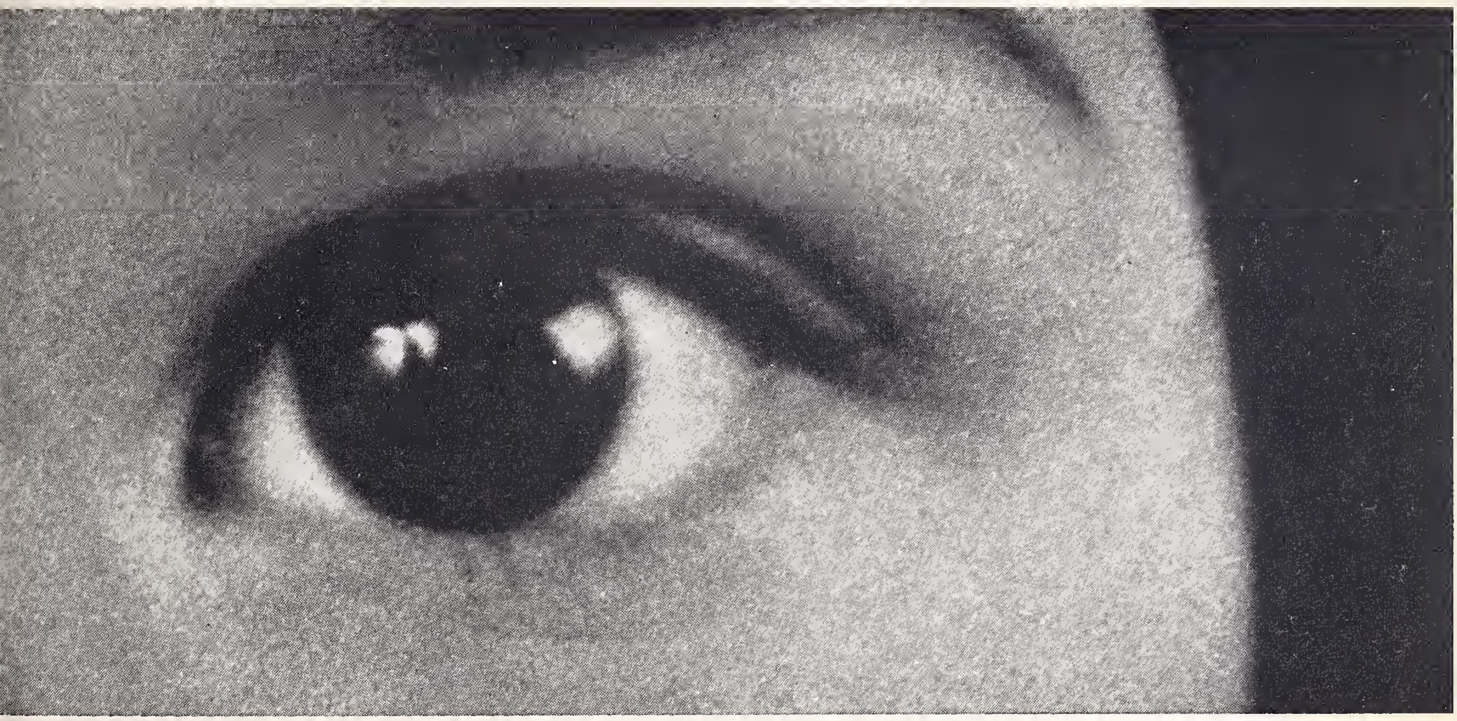
1. Zimmerman, F. T., and Burgemeister, B.: *Arch. Neurol. & Psychiat.* 72:720, 1954.
2. Zimmerman, F. T., and Burgemeister, B.: J.A.M.A. 157:1194, 1955.
3. Zimmerman, F. T.: *Arch. Neurol. & Psychiat.* 76:65, 1956.



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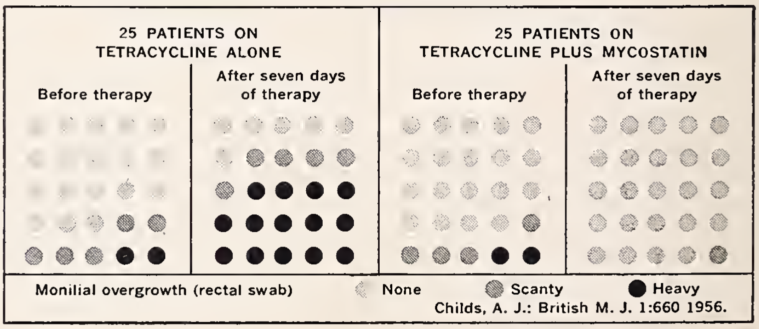

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Original Contributions

Thrombosis and the Blood-clotting Mechanism

CHARLES A. OWEN, JR., M.D.
Rochester, Minnesota

ONE OF THE MAJOR unsolved problems in the study of peripheral vascular disease is the explanation of thrombotic and embolic occlusion. It is not sufficient merely to propose that arteriosclerotic narrowing of a vessel's lumen or the slowing of blood flow explains thrombosis. Some persons who apparently have normal vessels experience occlusion idiopathically or, curiously enough, about the seventh day after an operation. Thorough analysis of the clotting mechanism of the blood in such cases might be profitable.

TABLE I. SCHEME OF BLOOD CLOTTING

$$\text{Platelets} + \text{AHG} + \text{PTC} + \text{PTA} \xrightarrow{(1)} \text{Plasma thromboplastin} + \text{Ca}^{++} + \text{Labile factor} + \text{Stable factor} + \text{Stuart factor} \xrightarrow{(2)} \text{Activated thromboplastin}$$

$$\text{Activated thromboplastin} + \text{Prothrombin} \xrightarrow{(3)} \text{Thrombin} \begin{cases} + \text{ Fibrinogen} \xrightarrow{(4)} \text{Fibrin} \\ + \text{ Antithrombin} \xrightarrow{(5)} \text{Metathrombin} \end{cases}$$

$$\text{Fibrin} + \text{Fibrinolysin} \xrightarrow{(6)} \text{Soluble degradation products of fibrin}$$

Because studies of abnormalities of blood coagulation have been limited traditionally to the hemorrhagic diatheses, little is known of the mechanisms contributing to thrombosis. In fact, most of the methods for study of clotting are developed about the principle of maximal acceleration of clotting *in vitro* to reveal any suggestion of retardation in one or another of the steps in clotting. As a result, if clotting is accelerated in certain thrombotic states, present methods are not likely to discover the fact.

Acceleration of the clotting cycle is theoretically possible and might well lead to intravascular thrombosis. If one adds oxalate to freshly drawn, normal human blood and removes the erythrocytes by brief centrifugation, leaving the platelets suspended, this plasma will clot in about 100 seconds at 37° C. on addition of a solution of a salt of calcium. How are these 100 seconds used up in the clotting mechanism? A scheme of clotting is necessary to arrive at an answer (Table I). The first four steps shown in the table are apparently essential to clotting; steps 5 and 6 have to do with quenching the reaction.

As just indicated, the first four steps in the clotting of normal human plasma at 37° C. occupy about 100 seconds. If the plasma thromboplastin that is formed physiologically in step 1 is replaced with tissue thromboplastin (the functions of these two types of thromboplastin are apparently identical), the plasma will clot in twelve seconds. This procedure, of course, is what is done during the Quick "prothrombin time" test performed with an extremely active extract of brain. Thus, elimination of the first step of clotting shortens the clotting time of the plasma from 100 to twelve seconds. Therefore, the complex interactions between platelets and the various hemophilic factors, namely antihemophilic globulin (AHG), plasma thromboplastin component (PTC) and plasma thrombo-

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From the Section of Clinical Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

plastin antecedent (PTA), occupy seven-eighths of the entire clotting process. By contrast, the second, or co-thromboplastin, step takes about six seconds, and steps 3 and 4 take the remaining six seconds.

If one is permitted to extrapolate these *in vitro* data to clotting *in vivo*, it becomes obvious that minutes to hours normally are required for the generation of plasma thromboplastin. Anything that might accelerate this first step, such as breakdown of platelets, or might eliminate this step, as the introduction of tissue juices into the blood stream, might induce clotting faster than the protective mechanisms—antithrombin (step 5) and fibrinolysin (step 6)—could function. Thus, thrombosis might be considered as the product of accelerated clotting *in vivo*.

Of course, the term "accelerated" presumes that clotting is occurring normally within the blood vessels, even if extremely slowly. There is reasonable likelihood that this does happen. The various clotting proteins that have been studied, namely, antihemophilic globulin, labile factor, stable factor, prothrombin and fibrinogen, all leave the blood stream at a much faster rate than do the major protein components of the plasma. Thus, negligible AHG remains in the blood of a hemophiliac patient twenty-four hours after a transfusion of fresh blood or plasma; the other clotting factors disappear rapidly after their source, which is the liver, has been removed from animals. It is unlikely that these clotting proteins are merely being metabolized in a nonspecific manner; it appears more reasonable that they are being consumed by clotting.

It is difficult, at first, to accept this hypothesis (by no means new) that clotting occurs continuously in the body, because one cannot find fibrin, which is the evidence of clotting, within the peripheral vessels or the lungs. However, fibrinolysin, which is the potent clot-dissolving enzymatic system of the blood, is presumably the scavenger that prevents traces of clotting from becoming thrombosis.

It may not be wise to condemn the theory of continuous intravascular coagulation just because fibrin cannot be seen. A classic experiment is the extremely slow injection of thromboplastin into the blood stream of an animal. When such a procedure is correctly performed, it is possible to convert the animal's plasma to serum, including complete loss of fibrinogen. Despite this, postmortem identification of fibrin is difficult or impossible.*

The well-recognized prophylactic and therapeutic effects of heparin and of dicoumarin derivatives furnish suggestive evidence that artificial retardation of clotting may compensate for a state of hypercoagulability. Further, the more rapid dissolution of an established clot during anticoagulant therapy suggests that a delicate balance exists between the formation and the lysis of fibrin that can be tipped in favor of the latter process by depressing the former.

Unfortunately, few factual data are at hand to support these various conjectures. It appears unlikely that much improvement in this situation will occur until new laboratory methods are designed that are directed at accelerated rather than at retarded clotting systems.

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*The obvious human analogy is the hemorrhagic tendency that is associated at times with abruptio placentae and that is inaccurately described as "afibrinogenemia." Since the loss of fibrinogen is secondary to coagulation of the blood, which in turn is the result of introduction of thromboplastin into the blood stream, the term "thromboplastinemia" more nearly describes this coagulation state.

BOOKLETS PUBLISHED FOR PATIENTS ON SODIUM-RESTRICTED DIETS

Three new booklets prepared especially for adult heart patients placed on sodium-restricted diets by their physicians have been published by the American Heart Association.

The booklets are available to physicians for distribution to their patients. They are also being made available to patients by local Heart Associations on a physician's prescription. Copies may also be obtained by nurses, nutritionists, dietitians and hospital and nursing home staffs.

The new booklets were prepared under the direction of a committee representing four agencies in addition to the American Heart Association. They are the U. S. Public Health Service, the American Dietetic Association, the Council on Foods and Nutrition of the American Medical Association, and the Nutrition Foundation.

Each booklet is designed to help patients follow the physician's prescription for a different degree of sodium restriction: strict, 500 mg.; moderate, 1,000 mg.; and mild. Within each booklet, there is a choice to be indicated by the physician, of three different calorie levels, 1,200 calories, 1,800 calories and unrestricted. Food lists enable the patient to vary his meals as he becomes familiar with his basic diet plan. Weight watching, meal planning and problems of the family cook are discussed and practical suggestions on eating out and shopping for food are given.

Diet, Physical Activity and the Serum Cholesterol Concentration

HENRY LONGSTREET TAYLOR, Ph.D.
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MORRIS[1] has reported that, in England at least, coronary heart disease occurs less frequently in physically active men than in their sedentary contemporaries. This finding has been confirmed by Brown et al[2] in a survey of men in the seventh decade who happened to be on the lists of eleven practitioners in Birmingham, England. These reports make the effects of physical activity on the level of serum cholesterol of some interest.

There are a number of factors which are known to influence the level of serum cholesterol, the most important being diet and sex hormones. Since exercise with its demands for calories influences the diet, it will be useful to discuss in detail the dietary factors which are known to be important in the regulation of cholesterol metabolism before taking up the specific effects of exertion.

Of the several dietary parameters which might affect cholesterol metabolism in man, the caloric balance,[3-5] the quantity of dietary fat[6-9] and the type of fat consumed[10-13] have been shown to be important factors affecting the level of serum cholesterol concentration. It has been customary for experimental investigators in this field to study the effects of alteration in the proportion of fats in the diet or to vary the total number of calories consumed, holding the energy expenditure and the amount of physical activity reasonably constant. Experiments of this kind and the study of the relationship between serum cholesterol levels and the composition of the habitual diet in population groups have led to the conclusion that when the quantity of fat is varied in ordinary human diets without changing the proportions of the several kinds of fatty acids in the fat, the percentage of calories derived from fat is the important factor in controlling the serum cholesterol concentration. If one holds the number of calories derived from fat constant along with the caloric expenditure and then feeds subjects fats which contain variable amounts of linoleic acid, it is found that fats with large quantities of linoleic acid depress the serum cholesterol concentration and fats which are made up of large proportions of saturated fatty acids produce increases in serum cholesterol concentration. This relationship is sufficiently constant to make it possible to predict with considerable precision the change in serum cholesterol concentration which will be produced by a change in either the amount and/or type of fatty acids in the diet.[13]

The caloric balance is an important factor which can have marked effects on the serum cholesterol concentration. A negative caloric balance will produce a fall in the serum cholesterol concentration,[3] while on the other hand, a positive caloric balance will produce an increase in serum cholesterol concentration.[4,5] It also follows that calorie equilibrium must be maintained if relative stability of the serum cholesterol concentration is to be maintained.

It is against this background of rather complex nutritional control of serum cholesterol that one must assess the effects of exercise.

The acute effects of exercise were studied in the Laboratory of Physiological Hygiene some time ago.[14] The results are summarized in Table I. In series A and B, cholesterol determinations were carried out in duplicate before and two hours after a breakfast which included eggs and heavily buttered toast. The value obtained after breakfast was subtracted from that obtained before breakfast and the difference is recorded in the

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From the Laboratory of Physiological Hygiene, The School of Public Health, University of Minnesota, Minneapolis, Minnesota.

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table. Series A differed from series B in that the serving of eggs contained 10 grams of cholesterol. In series C, breakfast was followed by moderately vigorous physical activity (walking on the treadmill or working in the garden). The fat-loaded meal in series A and B was followed by a small but significant rise in the serum cholesterol concentration while in series C the exercise resulted in a definite decrease. Combining the data, we can say that the effect of exercising after the fat-loaded meal was to reduce the serum cholesterol concentrations by 8 mg. per 100 ml. of serum. This result is statistically highly significant since the standard error is only ± 2.25 mg. per 100 ml. of serum.

TABLE I. THE EFFECTS OF EXERCISE ON THE CHANGE FROM THE BASAL STATE TWO HOURS AFTER A HIGH FAT BREAKFAST IN THE SERUM CHOLESTEROL CONCENTRATION ON MG. PER 100 ML.

Series	Post Meal Conditions	N.	Mean (Δ)	S.D. (Δ)	S.E. (Δ)
A	Sedentary	51	4.92	±11.72	±1.66
B	Sedentary	73	3.95	±11.74	±1.37
C	Exercise	15	−3.93	±12.95	±3.34

Experimental evidence of the effects of changing the level of daily exercise was provided by Mann and his co-workers[5] in the Harvard School of Public Health. These investigators examined the effects of increasing the calorie requirements of three men from 3,000 to approximately 6,000 calories a day by adding an intensive regimen of physical activity. The 3,000 additional calories were eaten in the form of carbohydrate; no additional fat was provided in the diet. The physical activity was continued at a high level for four weeks at which point it was discontinued without changing the daily ration of 6,000 calories of food. Table II shows the results obtained by Mann and his co-workers. During the period of increased physical activity, there was no change in the serum cholesterol concentration. The period of excess calories clearly demonstrated that a positive caloric balance results in an increased serum cholesterol concentration. Similar results have been obtained in this Laboratory.[4] Mann and his colleagues have interpreted this to mean that exercise prevented the increase in serum cholesterol during the activity period.

The effects of exercise in this experiment were not as clear-cut as one might like since the subjects were not able to burn up all the calories provided in the diet and consequently gained weight. Thus, there was a situation which, other things being equal, would be expected to result in an increase in serum cholesterol present. On the other hand, adding to the daily diet 3,000 calories in the form of carbohydrate resulted in a decrease in the percentage of calories derived from fat, a change which one would expect to result in a decrease in serum cholesterol under conditions of constant physical activity.

TABLE II. DATA OF MANN ET AL.[5] ON THE EFFECTS OF INCREASING THE DAILY PHYSICAL ACTIVITY BY 3000 CALORIES

Item	Control Period	Exercise Period	Excess Cal. Period
Time	2 weeks	4 weeks	4 weeks
Cal./day	2934	6000	5740
Fats as % cal.	52	26.1	27.4
Body weight	183	188.5	199
Serum cholesterol	180	178	230

In the Laboratory of Physiological Hygiene the effect on the serum cholesterol concentration was studied in the presence of several separate diets. The experiments were carried out in a standard situation and one of these experiments has been reported in detail.[15] In each case, the experimental design included a sedentary control period of two weeks. This was followed by a training period usually lasting a week or so in which the men accustomed themselves to walking for a period of two hours on the motor driven treadmill set at a grade of 10 per cent and a speed of 3.5 miles per hour. This activity required 1,200 calories a day and was maintained for not less than two weeks in addition to the training period in each experiment. The activity period was followed by a second sedentary period lasting from ten days to two weeks in which the diet was identical to that in the control period. Particular attention was paid to the maintenance of a constant body weight.

At the end of each period, blood was drawn on two successive mornings before breakfast. Each sample was analyzed in duplicate for total cholesterol and for the cholesterol associated with α and β lipoprotein. Total fats, non-esterified fatty acids, and phospholipids were analyzed to complete the picture.

In each experiment, 300 calories a day were allowed for calories that would be consumed by metabolism required by activities that would have been undertaken in the two hours devoted to treadmill work. The caloric cost of the exercise was then payed off with approximately 900 calories of food. The fat content of the food used to pay for the exercise was the item which was varied. In each case, great care was taken to ensure that the relative proportions of saturated to unsaturated fats remained constant in both the control period and the exercise period. Therefore, the principal variable here is the per cent of calories derived from fat. In the first experiment, the proportion of fat derived from calories in the food added in the exercise period was identical to that used in the control period. This resulted in the proportion of calories derived from fat being held constant in the three periods but the total amount of fat consumed by the men was increased during the exercise period. In the second experiment, the additional calories were 100 per cent fat, thus raising both the total fat ingested and the proportion of calories derived from fat during the exercise period. And finally in the third experiment, the added calories were 100 per cent carbohydrate, thus decreasing the per cent of calories derived from fat and holding the total amount of fat constant. The principal findings along with the dietary conditions are presented in Table III.

TABLE III. THREE EXPERIMENTS ON THE EFFECT OF INCREASING THE DAILY PHYSICAL ACTIVITY LEVEL ON THE SERUM CHOLESTEROL CONCENTRATION

(For explanation see text)

Exp. No.	No. of Subjects	Control Period			Exercise Period Additional Calories			Exercise Period Total Calories			Body Wt. △ in Pounds	Mean Serum Cholesterol mgms./100ml.		
		Total Cal.	Fat Grams	Fat % Cal.	Exercise Cal.	Fat Grams	Fat % Cal.	Total Cal.	Fat Grams	Fat % Cal.		Control	Exercise	Recovery
1	9	3250	155	43	930	46.0	44	4190	201	43	+0.60	158.3	159.8	161.5
2	5	3200	143	40	920	102	100	4120	245	53	+0.2	196.6	198.1	198.5
3	7	3200	145	41	900	0	0	4100	145	31.8	+0.35	199.5	181.1	195.8

It will be observed that increases of 46 and 100 grams of dietary fat a day were tolerated by the men during the exercise period without any appreciable increase in the serum cholesterol concentration. Elsewhere,[13] it has been shown that under conditions of constant caloric intake and fat composition, the percentage of the total calories derived from fat is the important controlling factor. If exercise did not modify the effects of diet, it would be predicted that the serum cholesterol concentration would remain unchanged in Experiment One, increase in Experiment Two and decrease in Experiment Three. In actual fact, it was predicted in Experiment Two on the basis of a prediction equation[13] derived from experimental work in which the composition of the diet was changed holding physical activity and total caloric intake constant that the serum cholesterol concentration would increase 28 mgs. per 100 ml. of serum in the group of men studied. Keys et al[13] did not study diets which contained as much fat as this, so the accuracy of the predicted value may be in some doubt, but there can be no question that increasing the percentage of calories derived from fat from 40 to 50 would be expected to result in a demonstrable increase in serum cholesterol concentration. We may conclude that within the range of fat content of diets studied, exercise allows a man to handle a large increase in the fat transport load without any comparable increase in serum cholesterol concentration and that it can alter the expected response of the serum cholesterol to alterations in the diet.

It is useful to regard the lipoprotein cholesterol complex as a vehicle for the transport of fat from one important metabolic site to another. Anderson, Lawler and Keys[4] have proposed that the observed variations in cholesterol concentration which result from dietary manipulation can be explained by the relationship between the fat transport load and the available fat transport, that is, lipoprotein cholesterol concentration per unit of circulation. The fat transport from gut to liver to fat depot was suggested as the general route involved.

Current concepts regarding the mechanism of fat transport are emphasizing two general pathways for transport.[16,17] One concept focusses on the lipoproteins, the other on non-esterified fatty acids (NEFA) but these are not viewed as mutually exclusive. It is, however, desirable to consider the possible relative importance of these two mechanisms of fat transport in various situations. In the first stage of fat metabolism, the absorption of fat from the gut and transport into the central blood pool, neither NEFA nor lipoprotein transport may be very important, the newly added lipid being primarily in the form of a suspension of chylomicrons in this phase of post-prandial lipemia.

There is, of course, much support for the idea that the fats are extensively hydrolyzed in or in close proximity to the gut wall and are then reassembled as neutral fat and are transported as such in the chylomicrons that characterize the lipemic phase. And there is little doubt that much of the absorbed fat is temporarily deposited as such in the liver. It is with the subsequent events, the transport of fat from the liver to the tissues and the mobilization of fat from depots elsewhere, that we are mainly concerned.

There is good evidence that under some conditions, at least, fat transport in the lipoproteins is very important. For example,[18] infants suffering from the general protein deficiency represented in kwashiorkor have grossly enlarged livers loaded with fat and very low levels of blood cholesterol from which we infer similarly low levels of lipoproteins in the blood. When such infants are treated simply by feeding proteins or even only a mixture of amino acids, the liver very rapidly loses its fat, the subcutaneous fat depots are repleted and in the process the blood serum cholesterol may rise in a few days from a pre-treatment value of 50 mg. per 100 ml. to one of 250, only to decline to more normal values in the region of 100 when this rapid transport of fat from liver to tissue ceases. The inference is that lipoprotein is importantly involved in this transport of fat.

On the other hand, large movements of fat from the depots to working muscles occur with no corresponding rise in cholesterol or lipoprotein concentration in the blood and there may even be a fall. We observed[19] that when young men are performing 1,100 calories of work a day and are fed only a diet of 1,010 calories of carbohydrate, the serum cholesterol concentration fell to 88 per cent of the control value in spite of the fact that large amounts of fat were clearly being transported from the depots to the sites of combustion.

Such evidence as the latter points to a second important mechanism of fat transport, one that would be capable of following large changes in metabolic requirements quickly. The second mechanism for fat transport which is currently being postulated is of this general type. It has been demonstrated that the non-esterified fatty acids are rapidly mobilized when the carbohydrate supplies are inadequate;[17,20,21] that it can account for as much as 70 to 90 per cent of the metabolism of cardiac muscles[17-20] and that it is mobilized from fat depots for utilization in other tissues.[20] Unfortunately no observations on non-esterified fatty acids were made in the semistarvation experiment referred to above but the evidence makes it a reasonable hypothesis to suggest that the non-esterified fatty acids played an important role in the large fat transport that must have taken place.

Let us for a moment return to the hypothesis that the lipoproteins play an important role in transport of fat from liver to fat depot or tissues. If this hypothesis holds, then any increase in fat load to be transported over this route without an increase in the circulation rate will result in an increase in the lipoprotein concentration. A decrease in the fat load with no change in circulation will result in a decreased lipoprotein concentration. Conversely, if the circulation is changed with no change in fat load, the concentration of lipoprotein will be inversely related to the changes in circulation. Finally, the changes in lipoprotein concentration will be reflected in the changes in serum cholesterol concentration provided the lipoprotein cholesterol complex remains reasonably constant in chemical composition.

In this form the theory appears to be consistent with the following observations:

1. When a positive caloric balance is present, the serum cholesterol concentration increases (increased transport of fat from liver to fat depots, only small changes in the circulation. Note that the excess calories may be principally carbohydrate).

2. The fall of cholesterol concentration in the serum cholesterol when a negative caloric balance is present (decreased fat transport from liver to fat depots, some decrease in circulation, the decrease in serum cholesterol is relatively small).

3. The increase in serum cholesterol concentration when the proportion of fat in the diet is

increased, caloric intake remaining constant (increased fat transport from liver to depots, no change in the circulation).

4. The relative stability of the serum cholesterol concentration when men are in caloric equilibrium on a diet of constant composition (transport load constant, circulation constant).

5. The decrease in serum cholesterol when the proportion of fat in the diet is reduced (a reduced fat load transported from liver to depots, no change in circulation).

In the exercise experiments reported here, the rate of metabolism is quite large during the actual work. The average oxygen intake is 2.2 liters per minute which means 10 to 11 calories a minute depending on the respiratory quotient (R.Q.). At an R.Q. of 0.85, approximately 5 calories a minute must be supplied by fat. It is reasonable to assume that such calories are supplied by either fat present in the muscle before exercise begins or fat transported to the muscle during exercise probably in the form of non-esterified fatty acids. Experimental work[22] in this Laboratory indicates that in steady state exercise lasting one hour the non-esterified fatty acids are close to the exercise starting level. But when work stops the concentration of the non-esterified fatty acids increases, on the average, 0.5 milleequivalents per liter with the first ten minutes and that this increase disappears after thirty to forty minutes. This strongly suggests that the rate of production is increased during exercise and that the piling up of non-esterified fatty acids in the blood is the result of a continuing production at a high level coupled with a decrease in utilization.

The implied conclusion resulting from Experiment One[15] that the lipoprotein cholesterol complex took part in transporting fat to the muscles in exercise appears to be incorrect in the light of Experiment Two. A more acceptable hypothesis would appear to be that the non-esterified fatty acids provided much of the fat transport during exercise. The findings presented here would appear to be consistent with the hypothesis that the lipoprotein cholesterol transport system can be largely bypassed during exercise. In the experiments under consideration, the exercise took place in a comparatively short time. If one is to judge by the slow increase in the cholesterol concentration after imposing a heavy fat load without changing the caloric requirement, fat transport by the lipoprotein cholesterol system is a slow but probably continuous process. The circulation between liver and skin or other fat depots is probably not much influenced by such a short intensive bout of exercise. The results of Experiment Three can be reconciled to the view presented here if one assumes that the exercise consumed enough fat to reduce the fat transport load from liver to fat depot while in Experiments Two and Three this was little affected because of the additional fat offered in the diet.

The view presented here suggests that the relation of exercise to meals may be important and that exercise at a lower rate spread over a longer period of time may have somewhat different effects.

Summary

It is clear that a number of questions have yet to be answered regarding the effects of exercise on the serum cholesterol concentration. Nevertheless, it can be stated that physical activity can allow man to increase the total quantity of fat ingested without a parallel increase in the serum cholesterol concentration and that exercise can alter the response of the serum cholesterol to dietary manipulation. The nature and extent of the alteration remain to be worked out. A theory which revolves around the transport of fat for storage and metabolic purposes is discussed in relation to new findings.

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Connective Tissue Changes Which May Be Related to Aging and Atherosclerosis

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THE PROBLEM of aging and atherosclerosis is assuming an ever-increasing prominence in this twentieth century society, for not only are the complications of atherosclerosis becoming painfully clear but the young and highly productive individuals of our population are being affected.

Most of the investigation into the problem of aging and atherosclerosis is in its initial stage. Our laboratories find themselves at the beginning of research into the tissue which appears to be altered perhaps as the initial event of the aging process. I wish to report to you on the work that has been completed in the study of connective tissue as it might pertain to these processes. The research opportunities to be found in connective tissue are vast and extremely exciting and they involve many diverse biochemical, physical chemical and immunochemical interests.

To orient thinking in regard to the aging blood vessel, it might be worthwhile to itemize information which is accepted by workers in this field.

Effects of Aging upon Vascular Tissue

(1) General
  (a) Internal elastic lamella becomes frayed, duplicated and replaced with collagen
  (b) Intima becomes thickened with collagenous tissue
  (c) Reduction in elastic properties of vessels occurs
(2) Coronary arteries
  (a) Intimal layer of coronary artery increases in size, particularly in males
  (b) Intimal thickening is greater in the left coronary artery than in the right
(3) Cerebral arteries
  (a) Intimal thickening and endothelial proliferation occur

Early in life, blood vessel changes which are ascribed to aging can be seen. The work of Moon and Rinehart[1] indicated that intimal thickening and fraying of the internal elastic lamella occur in infants a few months of age. A graph of the changes of the intima and the internal elastic lamella of the coronary arteries against the age of humans, revealed that these alterations occur early in life and reach their near maximum in the mid-thirties.

The arterial vessels of the body differ in fibrous protein composition.[2] The thoracic aorta is made up predominately of elastic fibers, but as the aorta passes through the diaphragm, a marked alteration in composition is observed. The abdominal aorta is made up of 70 per cent collagen and only 30 per cent elastin. The collagen-to-elastin ratio observed in the abdominal aorta is also seen in the peripheral vessels.

When a comparison is made between the fibrous protein composition of the arterial vessels and the early aging and atherosclerotic changes in the peripheral vessels, it is noted that the abdominal aorta, the iliacs and the coronary vessels with the greater percentage of collagen show the early changes, that is, in male patients, sixteen to thirty-nine years of age.[3]

To summarize what is known concerning the aging changes in the blood vessels, it appears that: (1) aging of an artery consists of an increase in connective tissue elements, particularly collagen; (2) vascular alterations occur early in life; (3) a predilection for certain vascular systems, such as the coronary, the abdominal aorta and the iliacs, exists and (4) the aging changes of the coronary tree are most commonly seen in the males.

Connective tissue is made up of ground substance, thick coarse bands of collagen, delicate elastic fibers and cellular elements consisting of fibroblasts, mast cells and histiocytes. The strength of connective tissue is derived from the high modulus of elasticity of the collagen fibers.

Collagen is formed by the arrangement of characteristic amino acids, particularly glycine,

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proline, and hydroxyproline, into a polypeptide strand by the fibroblast. Three polypeptide strands become laced into a spiral (which the physical chemists refer to as an alpha helix) and are held together by internal crosslinking.[4]

Because of the obvious importance of connective tissue (particularly the protein collagen) in the process of aging and because connective tissue is distributed in close association with muscles and joints, a technique by which connective tissue could be isolated from the remainder of the body was devised.[5] A plastic sponge was implanted in the subcutaneous tissue of the dorsum of laboratory animals, such as the rat, or along the left anterior chest wall of human volunteers. Through the interstices of the sponge, normal-appearing connective tissue developed. During the past four years, connective tissue obtained by sponge implantation has been studied in our laboratories and the following are some of the observations made.

Following the implantation of the sponge into rats, collagen is rapidly formed, appearing at the fifth to the seventh day, and increasing until the fourteenth to the twenty-first day. After twenty-one days of tissue growth, a sex difference in the amount of collagen was observed. In the female rat, the collagen content of the sponge twenty-one days after implantation was similar to that 300 days after implantation. The sponge implanted into the male animal continued to accumulate collagen even up to 300 days.[6] This difference could reflect the degree of metabolic inertness of collagen in the female or might indicate that the rates of synthesis and degradation of collagen equalled each other.

To determine the metabolic activity of collagen in the sponge-connective tissue of the rat, the rate at which lysine is converted to hydroxylysine was followed because hydroxylysine is a characteristic amino acid of collagen. It was evident that collagen obtained from the female rat was metabolically more active than that obtained from the male.[7] This suggested that in the female, collagen formation proceeded at a rate equal to collagen destruction whereas in the male, collagen formation occurred at a lower rate than in the female while collagen destruction proceeded at an even slower rate. Thus, the over-all effect was an increased collagen content in connective tissue obtained from the male but with no "net gain" of collagen in the sponge tissue from the female.

In addition to the variations in the rates of collagen formation and destruction, aging appears to influence the internal configuration of the collagen fiber as demonstrated by Baló and Banga[8] and by Verzár.[9] Rat tail collagen obtained from different-aged animals was exposed to a concentrated solution of potassium iodide and the contraction and relaxation of the fiber were followed. It was apparent that an accurate determination of animal age in months could be made by following the response of collagen to the solution of potassium iodide. "Young" collagen could be converted to "old" collagen by the exposure of the collagen fiber to dilute formalin solution. The effect of formalin on collagen is thought to occur by the addition of crosslinks between the three strands of polypeptides.[10]

Studies on the degree of acetic acid swelling of collagen obtained from Indians by sponge implantation indicated an apparent similar addition of crosslinks as a function of age. The swelling capacity decreases with age, and the effect of age is more apparent in the male than in the female donors.[11] The variation in swelling was interpreted to indicate a greater polymerization of the collagen in old than in young human volunteers.

To summarize the effects of age upon collagen: Fiber formation appears to be greater than fiber destruction, particularly in males. An increase in the strength and/or in the number of internal crosslinks of collagen occurs, that is, a form of accelerated polymerization.

Atherosclerosis

An atheroma appears to be a complication of the aging process. Some of the established facts concerning atheromatosis are:

(1) Endocrine
  (a) Predilection for males but occurs in females following the menopause
  (b) Accelerated by diabetes mellitus or oöphorectomy
(2) Vascular
  (a) Occurs in the intima
  (b) Patchy in character—localized at sites of bifurcation or sharp turns of a vessel
  (c) May be induced in a vessel as the result of the trauma of eddies or currents, that is, jet lesion
  (d) Appears to be accelerated by hypertension
(3) Metabolic
  (a) Increased lipids and elevated cholesterol concentration of the serum are associated with atherosclerosis in some patients

Considerable controversy has arisen concerning the morphogenesis of an atheroma. To examine the biochemical changes occurring in the aorta with atheromatosis, the normal intima, intima containing an early atheroma and intima containing an advanced atheroma were studied.[12] No significant increase in cholesterol or lipid concentration was observed in the early atheroma. The advanced atheromatous area of the male contained no significant lipid elevations other than cholesterol, whereas in the female, other lipids (phospholipid and neutral fat) were also significantly increased. The significant biochemical changes noted consisted of an increase in certain amino acids, that is, glycine and hydroxyproline, which characterize collagen, and of the binding of hexosamine to the collagen of the intima. Thus, it appears that localized fibrogenesis of the intima preceded the lipid atheromatous lesion, a fact often suggested by the pathologists.

Fibroblasts synthesize cholesterol from a tagged two-carbon acetate molecule *in vitro*. The rate of cholesterol synthesis in rat connective tissue was greater in the male than in the female. Examination of the effects of pretreatment of the animal with various sex hormones revealed that estrogen enhanced the rate of cholesterol formation in the connective tissue obtained from the rat. The only hormone which depressed synthesis rate in both sexes was the hormone of pregnancy, relaxin. A dichotomy of response to sex hormones was observed between the connective tissue slices and the simultaneously incubated liver slices. The hormone which appeared to affect connective tissue and liver in a similar fashion was estrogen in the male rat.[13]

The effects of exogenous cholesterol upon rabbit connective tissue were followed. Multiple sponge implants were made and the biopsy-connective tissue was removed at various time periods. It was apparent that while the animal was on a high cholesterol diet increases in cholesterol (both total and esterified), total phospholipid and neutral fat of connective tissue occurred. Upon the cessation of the cholesterol diet and the return of the animal to a diet containing only trace amounts of cholesterol, the tissue phospholipids decreased as did the neutral fat. However, the cholesterol concentration of the connective tissue continued to rise. It was noted that at 170 days the serum cholesterol had returned to its control value while the tissue cholesterol had mounted to a concentration of almost 4 grams per cent. Thus, almost a twenty-fold reduction in serum cholesterol value failed to influence the cholesterol concentration of the sponge connective tissue.[14] Examination of the sponge-tissues from the cholesterol-fed animals indicated that the connective tissue had the appearance of an atheroma or a xanthoma.

Thus, it can be concluded that the fibroblasts synthesize cholesterol and that a large amount of exogenous cholesterol accumulates in young connective tissue. The localization of cholesterol in connective tissue is a subject of current investigation in our laboratory.

Summary

Aging produces changes in connective tissue, one of the principal tissues of the artery. These changes consist of a greater production than destruction of collagen, which results in an accumulation of collagen in the human male and postmenopausal female and in the male rat. Collagen obtained from old humans has apparently undergone a polymerization process with the addition of crosslinks between strands of the collagen polypeptide strands. At the site of vascular stress, localized fibrogenesis occurs with the formation of young connective tissue. New connective tissue, such as that obtained by sponge implant, not only synthesizes cholesterol but has a strong affinity for exogenous cholesterol.

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Observations on the Turnover of Cholesterol in a Human Subject

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THIS ARTICLE will be concerned principally with the routes of cholesterol excretion in man. Its justification is the emergence of new data which may require revision of some of our concepts on this subject.

Historical Considerations

Until recent years, the commonly held picture of cholesterol catabolism was as follows: Blood cholesterol is excreted through the wall of the intestine into the lumen. There, by the action of bacteria, it is converted to a greater or lesser extent into other substances, the chief of which is coprostanol. These degradation products, along with some unchanged cholesterol, are eliminated with the feces. In 1943, Bloch, Berg, and Rittenberg demonstrated the conversion of cholesterol to cholic acid in the dog.[1] Their experiments were not designed in a way to permit evaluation of the quantitative importance of the bile acids in cholesterol catabolism.

The picture was drastically altered when Chaikoff, Siperstein, and their co-workers[2,3] showed that in the rat, the bile acids are the most important end-products of cholesterol metabolism. More recently, Siperstein and Murray[4] carried out an isotopic experiment on a human subject with a temporary surgical bile fistula, and their results have been interpreted as evidence that the bile acid route is, quantitatively, the most important pathway in man, as well. This interpretation seems to have received rather wide acceptance. See, for example, a recent review[5] in which seventeen references are cited in support of it.

We have been reluctant to accept the bile acids as the principal medium for the excretion of cholesterol in man. Our hesitancy is based on two considerations: (1) We have been unable to detect in feces anywhere near the quantities of bile acids that would be expected from the turnover rate of cholesterol.[6] (2) Human feces, even with the subject eating a low sterol diet, contain large amounts of unsaponifiable, digitonin-precipitable material, the source of which is obscure if it does not represent excreted blood cholesterol.

Plan of the Experiment

The points mentioned above might possibly be reconciled if one were to assume that the bile acids are altered so drastically by the intestinal flora as to be unrecognizable in the stools, and, further, that the fecal coprostanol is derived largely from the intestinal mucosa, without prior mixing with the cholesterol pool of the blood. To test these hypotheses, the experiment described below was designed. The general plan was to inject cholesterol labeled with carbon 14 intravenously into a normal human being on a diet low in sterols, and then fractionate the radioactivity in the stools. If the fecal coprostanol is not derived from the blood, its specific activity should be much lower than that of the blood cholesterol. If large amounts of bile acids are excreted in altered condition, this fact should present itself in the form of a sizable amount of radioactivity falling neither in the digitonin-precipitable fraction nor in the known bile acids.

Materials and Methods

The subject was a fifty-two-year-old healthy man who was kept on a metabolic ward for the duration of the experiment. He was fed a formula diet which provided 2015 calories per day and consisted of skim milk 6 cups, whole milk 5 cups,

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sucrose 50 gms., chocolate syrup 100 gms. (protein 96.1 gms., carbohydrate 250.4 gms., fat 78.2 gms., cholesterol 169 mg.).

The cholesterol for injection was labeled at carbon 4, and was obtained from the Nuclear Instrument and Chemical Corporation. Its specific activity was 5μ/milligram. It was homogeneous by chromatography on silicic-acid-Supercel.[7] The dose to be given was dissolved in 4 ml. of 95 per cent ethanol, which was added to 35 ml. of isotonic sodium chloride solution. This mixture was administered intravenously during a period of about five minutes.

For determination of the specific activity of the serum cholesterol, free and total cholesterol were extracted and precipitated as the digitonide, as in the Schoenheimer-Sperry method,[8] except that all volumes were increased appropriately. Cholesterol was recovered from the digitonide by the method of Schoenheimer and Dam.[9] The recovered material was dissolved in 10 ml. of toluene containing 0.3 per cent 2,5-diphenyloxazole, and counted in a coincidence type liquid scintillation counter, with an efficiency of about 70 per cent. The amount of cholesterol counted was determined colorimetrically on a 1 ml. aliquot of the toluene solution, after evaporation to dryness. The Liebermann-Burchard reaction, as ordinarily carried out in the Abell method,[10] was used. The 3 mg. of fluor contained in the aliquot was found not to interfere.

Red cell cholesterol was isolated as follows: The packed cells were hydrolyzed for three hours in five times their volume of 15 per cent potassium hydroxide in 95 per cent ethanol. An amount of water equal in volume to that of the hydrolysate was added, and the mixture was extracted three times with twice its volume of petroleum ether (b.p. 36° – 60°). The petroleum ether was removed by distillation, and the specific activity of the cholesterol determined, as noted above.

For the recovery of labeled lipids from the feces, the specimen was weighed and mixed thoroughly, and an aliquot was extracted with boiling ethanol and petroleum ether, as previously described.[6] When the residue from this extraction was subjected to hydrolysis with alcoholic KOH and extraction with petroleum ether (as described above for red blood cells), less than 1 per cent of additional radioactivity could be removed. Counting of the dry residue itself as a check on the efficiency of the extraction was not very satisfactory because of the large mass involved, and the resulting low observable counting rate. An attempt to test the method in this way indicated that about 5 per cent of the radioactivity was unextracted, but this figure is undoubtedly too high because no correction could be applied to eliminate the counts from naturally occurring radioactive potassium.

Since one of the prime objects of the experiment was to make a balance sheet of the radioactivity in stools, aliquots of the crude alcohol extract were evaporated to dryness and counted, either as infinitely thin or infinitely thick plates. Additional aliquots were subjected to saponification and extraction as described above, and the unsaponifiable matter was plated for counting. The digitonin-preciptable fraction was also counted as the infinitely thick digitonide. Some of it was recovered from the digitonide, and aliquots were weighed and counted in the liquid scintillation counter, for direct comparison with the blood cholesterol. The infinitely thick samples were counted with a thin window Geiger-Mueller counter, but many of the thin samples were counted in a windowless flow counter. Although factors were worked out for the interconversion of results obtained in these various ways, all of the important comparisons were based on values obtained by identical techniques.

Fecal bile acids were extracted and isolated chromatographically as previously described,[6] except that in certain instances the residue from the ethanol extract was dissolved in 70 per cent aqueous ethanol and extracted repeatedly with petroleum ether (b.p. 60° to 70°), in order to provide a better initial separation of the bile acids from cholesterol, coprostanol, and other neutral sterols, which are not efficiently extracted from aqueous suspension.

The dimensions of the Celite columns used for the bile acid separations were 1.8 x 30 centimeters. The stationary phase was 70 per cent acetic acid, and elution was accomplished with 150 ml., 300 ml., and 450 ml. of petroleum ether containing 0, 40, and 60 per cent isopropyl ether, respectively. The eluate was collected in 200 tubes, each containing 4.5 milliliters. Aliquots from the tubes were placed on stainless steel planchets and the radioactivity counted in a gas flow counter. The remainder of the eluate in each tube was titrated with 0.015 N sodium hydroxide.

Methyl deoxycholate was prepared for liquid scintillation counting by dissolving the deoxycholic acid eluted from the paper chromatogram, usually a quantity of about 0.5 mg., in 10 ml. of hot methanol containing HCl in a concentration of 0.28 N. The methanol-HCl solution was prepared by exposing the methanol to dry HCl gas.

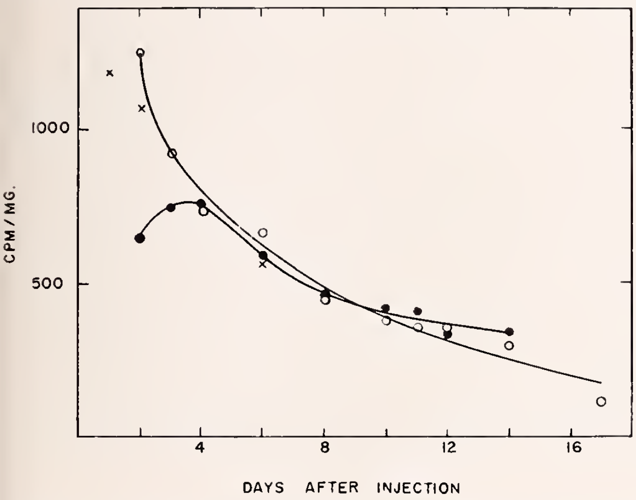

Fig. 1. Specific activity of blood cholesterol following intravenous injection of C^{14} labeled cholesterol.
○ Free cholesterol
● Esterified cholesterol
x Red cell cholesterol

Experimental

The subject was given 20 microcuries of C^{14}-labeled cholesterol intravenously. All stools were saved, and 20 ml. of blood were drawn daily. Figure 1 shows the decline in the specific activity of the free and esterified serum cholesterol, and of the red cell cholesterol. All of these fell quite close together during most of the experimental period. The specific activity of this pool declined by a factor of 2 between the fourth and tenth day following the injection.

Table I shows the weight of digitonin precipitable sterols isolated from the stools on the sixth, eighth, and tenth days after administration of the tracer, and a comparison of their specific activity with that of the total serum cholesterol on the same day. It is evident that the amount of endogenous sterols excreted by this patient was of the same order of magnitude as the values reported in the literature. Furthermore, the specific activity of these sterols corresponded to that of the blood a day or two earlier, providing proof that they came from the blood, without appreciable dilution from any other source.

Table II shows the distribution of radioactivity between unsaponifiable and bile acid fractions for the stool collected on the ninth day. Most of the cholesterol excretion products fell in the unsaponifiable fraction.

TABLE I. WEIGHTS OF DIGITONIN PRECIPITABLE STEROLS IN FECES, AND COMPARISON OF THEIR SPECIFIC ACTIVITY WITH THAT OF THE BLOOD CHOLESTEROL

	Days After Injection of Radioactive Cholesterol 6	8	10
Specific activity of total serum cholesterol, CPM/mg.	616	463	413
Specific activity of digitonin precipitable fecal sterols, CPM/mg.	665	583	462
Weight of sterols recovered from the digitonides, mg./day	621	976	635
Wet weight of stool, grams	79.7	128.0	80.9
Weight of aliquot extracted, grams	16.2	26.0	17.5
Weight of sterols actually counted, mg.	5.27	7.38	5.43

The counting was done in the liquid scintillation counter.

TABLE II. DISTRIBUTION OF RADIOACTIVITY BETWEEN UNSAPONIFIABLE AND BILE FRACTIONS FOR THE STOOL COLLECTED ON THE NINTH DAY

	Radioactivity in CPM	Per Cent of Total
Total ethanol extract	623,000	100
Unsaponifiable	450,000	72
Bile acid fractions	106,000	17
Unaccounted for	68,500	11

The counting was done in the windowless flow counter, at infinite thinness. Aliquots of several sizes were counted in each instance, to establish the fact that the counting rate was actually proportional to the size of the aliquot. The figure for the bile acids was obtained by adding the counts of the fractions which emerged from the column (Fig. 2). The nature of the compounds containing the radioactivity unaccounted for is unknown. They represent material which was not removed from the column with the solvents used.

TABLE III. RECOVERY OF UNSAPONIFIABLE MATTER FROM THE STOOL OF THE SEVENTH DAY

	Radioactivity in CPM	Per Cent of Total
Total ethanol extract	9500	100
Unsaponifiable	6700	70
Unsaponifiable, digitonin precipitable	5300	56

The counting was done with a thin window Geiger-Mueller counter, at infinite thickness, in dishes 2.5 cm. in diameter. The weight of material counted was always at least 140 mg., and the counts recorded above were corrected to the values that would have been obtained if all of the radioactivity present had been contained in a sample of that weight.

Table III shows the recovery from the stool of the seventh day, with the unsaponifiable matter divided further into fractions which did and did not precipitate with digitonin. Eighty per cent of the radioactivity in the unsaponifiable fraction was precipitable.

An example of the chromatographic distribution of radioactive and acidic compounds in the feces from the eighth day of the experiment is shown in Figure 2. Deoxycholic acid (peak D) was further purified by paper chromatography[11] and eluted from the chromatogram with ethanol. The ultraviolet absorption spectrum of the product thus obtained was identical with that of pure deoxycholic acid. The concentration was determined from the absorption maximum at 385 μ. Radioactivity of the methylated material was measured in the liquid scintillation counter. The specific activity was 590 C.P.M./mg., compared with 463 C.P.M./mg. and 583 C.P.M./mg., respectively, for the serum and fecal digitonin precipitable sterols of the same day. The specific activity of the fecal deoxycholic acid of the seventh day was 620 C.P.M./milligrams.

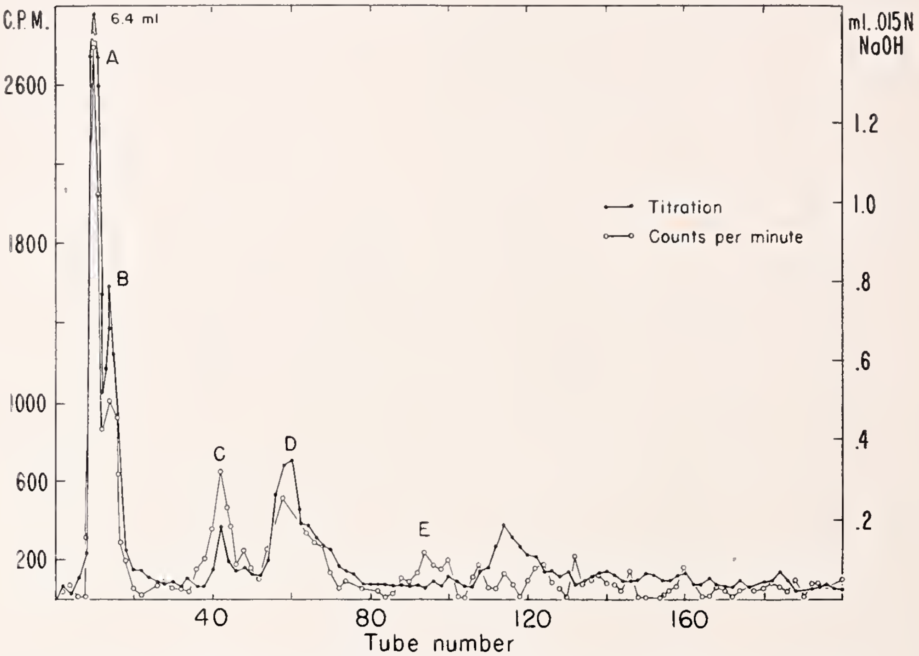

Fig. 2. Distribution of radioactive and acidic compounds in the bile acid fraction of feces eight days after the cholesterol-4-C^{14} intravenously.

A considerable amount of the radioactivity in peaks A and B (47 per cent) was found to result from a petroleum ether-soluble, digitonin-precipitable compound which was not removed by the initial separation. The remaining 53 per cent of the radioactivity was present as a non-digitonin-precipitable substance, soluble in alcohol. Some of the acidity in these peaks was non-radioactive fatty acid.

The amounts of deoxycholic acid excreted on the seventh and eighth days of the experiment were 24 mg. and 32 mg., respectively, accounting for less than 5 per cent of the total sterol excreted. The remainder of the bile acids had been sufficiently altered by intestinal flora so as to be no longer identifiable by the techniques used. It is quite probable that much of the radioactivity in the unaccounted-for category can be attributed to these altered bile acids or their derivatives.

Discussion

These results seem to indicate that in this individual, the bile acids played only a minor role in the excretion of cholesterol. An explanation is still required for the apparently opposite findings of Siperstein and Murray. Since both our conclusions and theirs are based on single experiments, individual variations may account for the discrepancies, and it is obvious that more work is needed. We wish to point out two features of their experiments, however, which may account for the differences:

(1) Their patient had a bile fistula, so that the normal reabsorption of bile acids was prevented. Under these circumstances, the turnover of bile acids might be expected to increase, and has, indeed, been shown to do so in the rat.[12] In their experiment, 39.6 per cent of the radioactivity administered was recovered in a period of fifty hours. We may calculate that the half-life of the body cholesterol with which the tracer was mixed was about sixty-eight hours, a much lower value than that commonly accepted for normal human subjects. Therefore, it appears likely that

the conversion of cholesterol to bile acids was accelerated under their experimental conditions.

(2) Their balance sheet of excreted radioactivity was based on a period of only fifty hours following administration of the tracer. Because of the time normally required for the passage of materials through the intestinal tract, a falsely low fraction of the radioactivity would be expected in stools collected during such a period.

An interesting conclusion may be drawn from the fact that the deoxycholic acid from our patient's stools had a specific activity approximating that of the serum cholesterol. Evidently, this deoxycholic acid was derived virtually entirely from cholesterol. No measurable quantity arose from unlabeled precursors, such as acetate, without going through the cholesterol pool.

Summary

(1) Carbon 14 labeled cholesterol was administered intravenously to a human subject on a diet low in sterols, and blood and stools were collected for a period of seventeen days.

(2) The stools were found to contain 0.5-1.0 gram of unsaponifiable, digitonin-precipitable material daily, the specific activity of which was equal to that of the blood cholesterol one or two days earlier.

(3) Fractionation of the radioactivity of the stools led to the following balance sheet: deoxycholic acid, 3 to 5 per cent; other compounds in the bile acid fraction, 13 per cent; unsaponifiable, 72 per cent, of which 80 per cent was precipitable with digitonin; unaccounted-for, 11 per cent.

(4) It was concluded that in our patient, the principal route of cholesterol excretion was the unsaponifiable fraction of the stools, with the bile acids playing a relatively minor rôle.

(5) The fecal deoxycholic acid was derived entirely from cholesterol.

Acknowledgments

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AGING AND ATHEROSCLEROSIS

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Vasomotion in Systemic Arteries, Small Vessels and Veins Determined by Direct Resistance Measurements

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LITTLE ATTENTION has been given the possibility that macroscopic vessels might contribute significantly to the maintenance and adjustability of resistance to blood flow across systemic vascular beds. These functions have been mainly attributed to the arteriol. Arteries and veins are relatively abundantly supplied with smooth muscle and autonomic nerve connections. When surgically exposed, they respond to various local physical and chemical stimuli with intense constriction. Despite this demonstrated potential, the details of their behavior under usual conditions are poorly understood. The obvious importance of artery and especially vein caliber changes to blood volume distribution, cardiac filling and water and salt transport across capillary membranes seemed to justify a detailed study of small and large vessel resistance responses to various stimuli. Partial findings of this study follow.

Methods

The methods employed were developed in Dr. M. B. Visscher's laboratory at the University of Minnesota and subsequently refined at the Northwestern affiliated V.A. Research Hospital in Chicago and at the Army Medical Research Laboratory at Fort Knox, Kentucky. Dr. Visscher suggested the study in 1951 and many of the ideas eventually incorporated into it were originally his.

Artery, vein and small vessel caliber changes in the foreleg of the anesthetized dog were inferred from resistance values calculated from directly measured levels of pressure and flow rate (Fig. 1). Foreleg blood flow rate was either controlled with a blood pump[*] or measured with a flow meter[**] interposed in the brachial artery. Pressures were measured at four sites along the length of the bed. These sites included the brachial artery just distal to the pump or flow meter, a foot pad small artery, a dorsal subcutaneous paw small vein and the cephalic vein at about the level of the elbow. Pressure gradients from brachial artery to cephalic vein, brachial artery to small artery, small artery to small vein and small vein to cephalic vein were calculated. Resistance values for the entire bed, arteries, small vessels (arteriol, capillary, venule) and veins were separately derived by dividing the respective gradients by the blood flow rate.

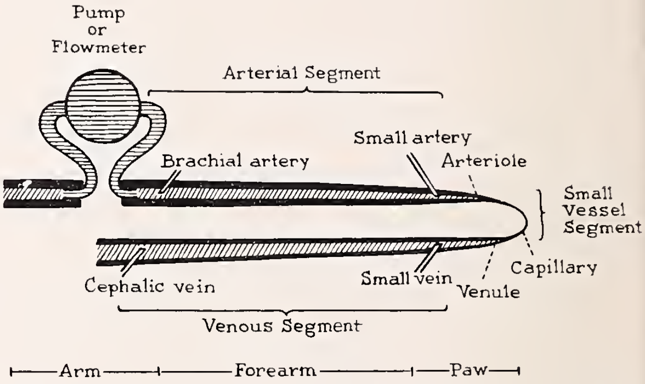

Fig. 1. Schematic drawing showing experimental arrangement for measurement of resistance to blood flow in arteries, small vessels and veins of dog foreleg.

Pressures were measured in the small artery and vein by entering 1 mm. sized vessels with 0.5 mm. glass or stiff plastic catheters[†] and threading them distally as far as possible (Fig. 2). The catheters, about 6 cm. in length, were advanced 4 to 5 cm. peripherally. It was necessary to manipulate the tip through valves in the case of the vein. The

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[*] Sigmamotor Pump Model T-6, Sigmamotor, Inc., Middleport, N. Y.

[**] Rotometer and Recording Unit No. 45, Clifford Wilson, 325 West 42nd Street, Indianapolis 8, Indiana.

[†] Albert E. Afford, Williams Ave., Barrington, N. J.

1 mm. vessels were entered either following surgical exposure or by needle puncture through the intact skin. In the latter case, a 21 thin walled needle‡ was directed peripherally and the catheter passed through it. The needle was then withdrawn. Since in their final positions the catheter tips completely occluded the vessel, the intraluminal pressures recorded§ were then those in distal collateral vessels. Small and large vein pressures were also studied in trained wakened dogs under local anesthesia and in wakened humans. Detailed descriptions of the methods employed have been published previously.[1,2]

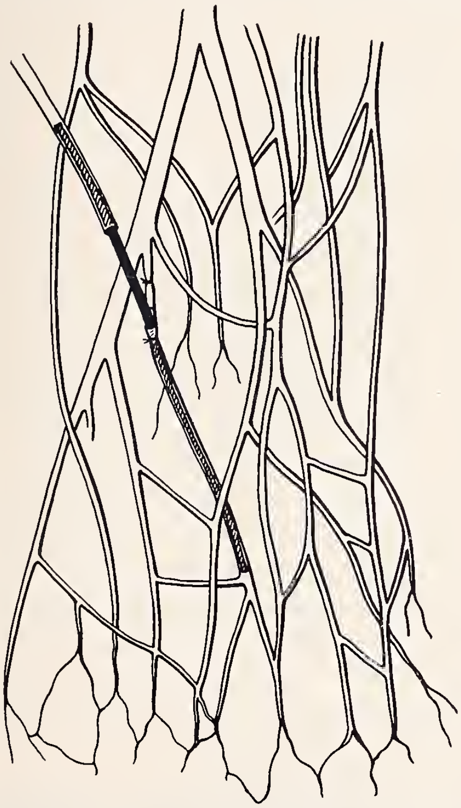

Fig. 2. Method used for measurement of pressure in dorsal limb subcutaneous small veins. Catheter completely occludes lumen of 0.5 mm. vein and pressure recorded is then that in distal collaterals. Vessel surgically exposed only at point of entrance of catheter and tied proximally. Catheter manipulated in retrograde direction 4 to 5 cm. Catheter may also be inserted through intact skin utilizing special thin wall needles. Same method employed for measurement of small artery pressure.

Results

General Anesthesia

General Considerations.—Small vein pressure exhibits cardiac and respiratory variations which resemble those in large systemic arteries.[1] The waves are not affected by venous compression but disappear upon arterial occlusion suggesting that they are transmitted from the arterial side. Though the mean pressure in a given vein remains quite constant with time it ranges from 8 to 28 mm. Hg. between different veins. This interindividual variation in pressure level likely is related to differences in venous resistance, blood flow rate and size of vessel catheterized between forelegs. The average small vein pressure value is about 14 mm. Hg. and this is to be compared to that of 4 mm. Hg. for the cephalic vein in the same leg. A positive small to large vein pressure gradient is always present. Foot pad small artery pressure averages about 75 mm. Hg. This level also varies from vessel to vessel likely for reasons similar to those listed for small vein pressure.

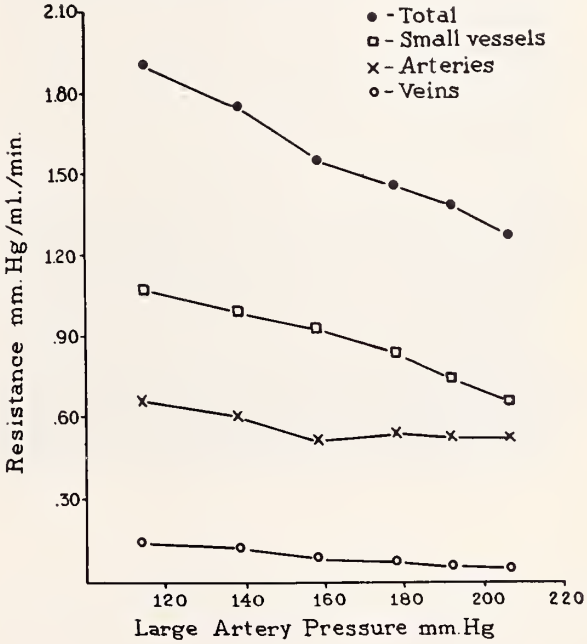

Fig. 3. Relation of total and segmental foreleg vascular resistances to brachial artery pressure. Anesthetized animal. Changes induced by varying foreleg blood flow rate with a pump inserted high in brachial artery. Extreme flow rates 56 and 163 ml./min. The values for small artery, small vein and large vein pressures at these flow rates were 75 and 125. 15 and 16, and 6 and 7 mm. Hg., respectively. Resistance variations represent homogeneous passive changes resulting from change of transmural pressure.

Homogeneous Passive Resistance Changes.—Figure 3 shows that elevation of vessel pressures by varying blood flow rate with the brachial artery pump is associated with resistance decreases in all segments.[2] That the resistance variations likely represent changes in caliber is indicated by

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‡Bishop Blue Label Hypodermic Needles, J. Bishop and Co. Platinum Works, Malvern, Penna.

§Any manometer with a volume displacement of 0.05 mm.^{3}/100 mm. Hg. or less is adequate.

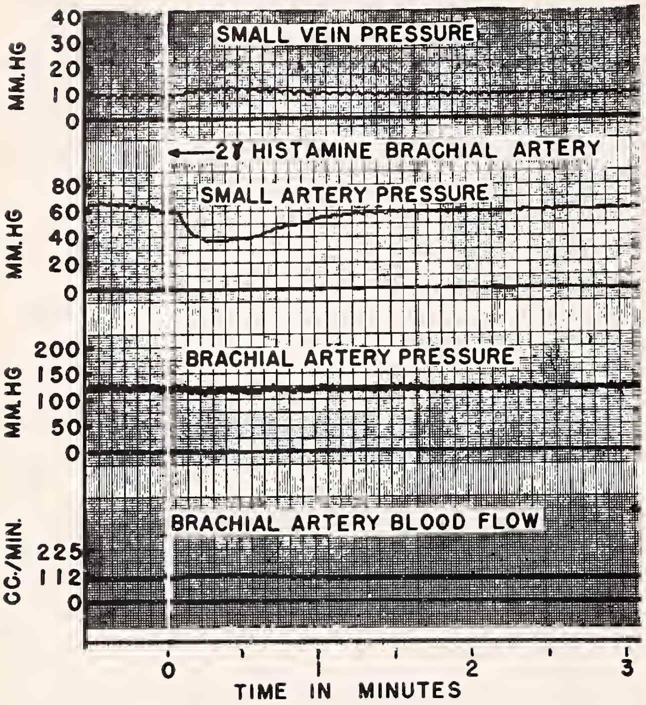

Fig. 4. Effect of histamine phosphate upon brachial artery, small artery and small vein pressure and blood flow rate in foreleg of pentobarbitalized dog. Flow rate measured with flowmeter interposed high in brachial artery. Histamine injected instantaneously into brachial artery just distal to flowmeter. Cephalic vein pressure, not shown here, did not change.

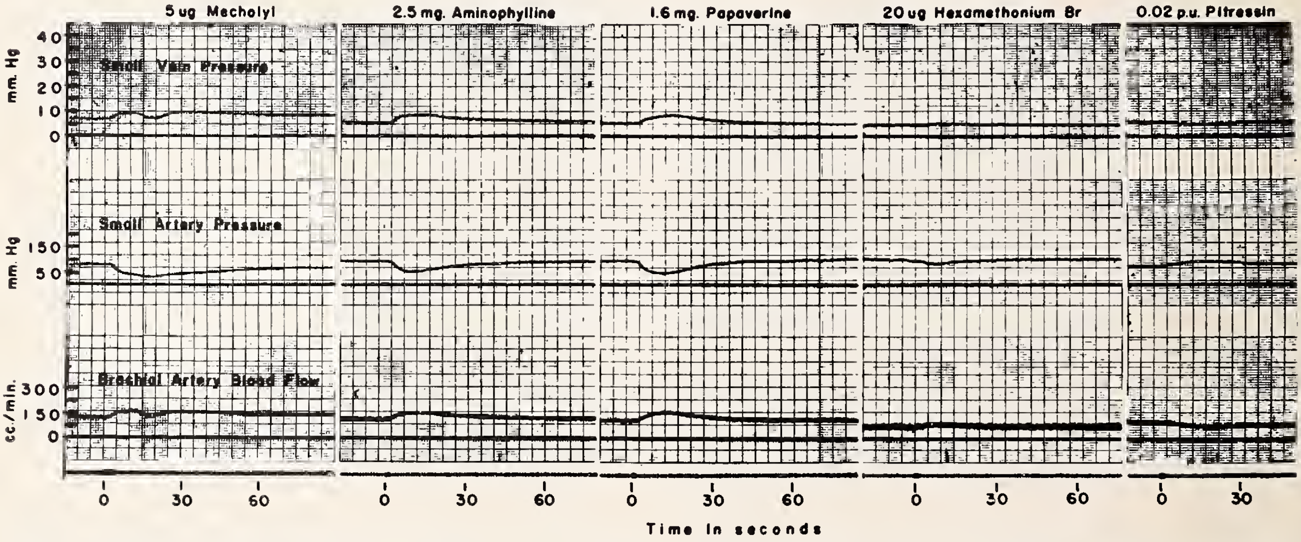

Fig. 5. Effect of instantaneous brachial artery injection of various substances upon foreleg blood flow rate and small vessel pressures. Brachial artery and cephalic vein pressures did not change significantly.

the fact that the variations are greater than might be expected from dynamic changes in viscosity alone. Resistances decrease because intraluminal and hence, transmural pressures increase, thereby passively increasing vessel calibers. On the other hand, lowering pressures by decreasing blood flow rate results in a resistance increase in each segment due to passive constriction.

Homogeneous Active Resistance Changes.—Small brachial artery doses of acetylcholine, acetyl beta metha choline, histamine, aminophylline, papaverine, ethyl alcohol, hexamethonium, pitocin and priscoline greatly decrease small artery pressure, slightly increase small vein pressure and increase blood flow rate (Figs. 4 and 5). Brachial artery and cephalic vein pressures do not change.[3,4] One of these, acetylcholine, has been studied in detail (Fig. 6). Blood flow rate was maintained constant with the pump while acetyl choline was constantly infused into the brachial artery. Under these conditions large artery, small artery and small vein pressures fall. Calculated resistance decreases in each segment. The active nature of the caliber increases is established by the fact that they occur in the presence of falling intraluminal pressures. This same response is usually observed in the rewarming period following cold exposure. Denervation also actively dilates the small vessel segment. The addition of intra-arterial phentolamine induces further active small vessel dilatation and sometimes active artery dilatation.

Intra-arterial injections of commercial epinephrine, 1-epinephrine (Fig. 7) and 1-norepinephrine cause large increases of small artery and vein pressures. Blood flow rate decreases and large artery and vein pressures remain unchanged. Pitressin, angiotonin and ergotamine cause the same changes except that small vein pressure remains unchanged or falls slightly (Fig. 5). Commercial epinephrine and 1-norepinephrine have been studied[5] when administered by constant infusion with blood flow rate maintained constant (Fig. 8). Norepinephrine elevates brachial artery, small artery and small vein pressures. Resistance increases in each segment. Attesting to the active nature of the caliber decreases is the fact that all calibers decrease in the presence of increasing intraluminal pressures. This same response usually

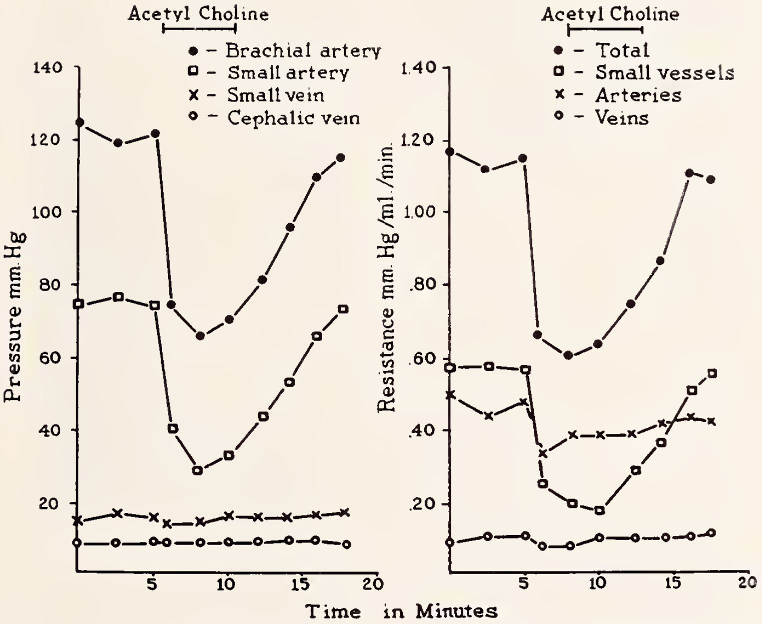

Fig. 6. Effect of acetylcholine upon vascular pressures and segmental vascular resistances in foreleg of pentobarbitalized dog. Blood flow rate maintained constant at 100 ml./min. with blood pump interposed high in brachial artery. Acetylcholine constantly infused into brachial artery just distal to pump at rate of 30 γ/min. Resistance changes represent homogeneous active dilatation.

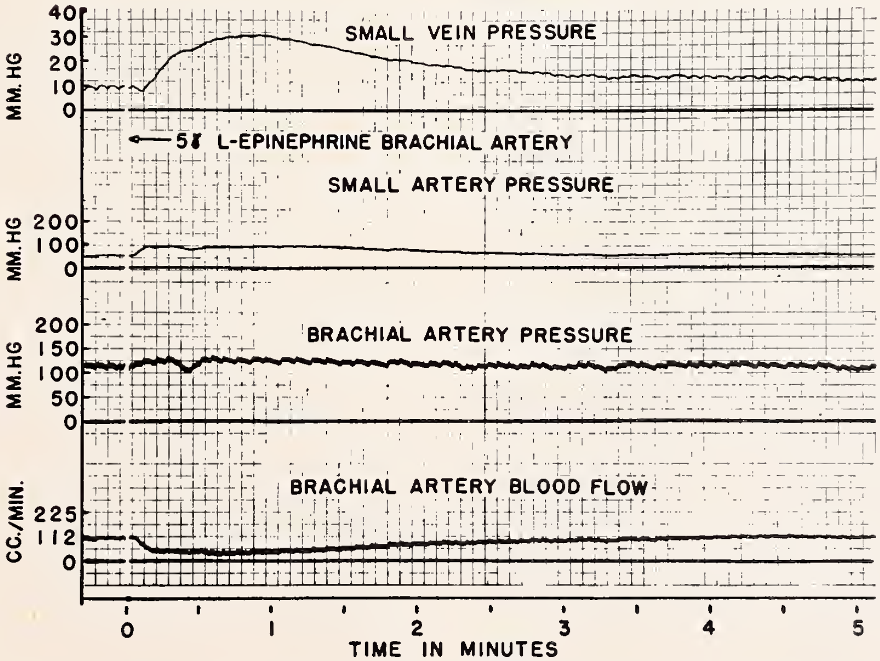

Fig. 7. Effect of instantaneous brachial artery injection of 1-epinephrine bitartrate upon small and large vessel pressures and blood flow rate in foreleg of pentobarbitalized dog. Cephalic vein pressure did not change.

occurs during cold exposure. Bilateral vagotomy also regularly causes active small vessel constriction associated with less regular active artery constriction.

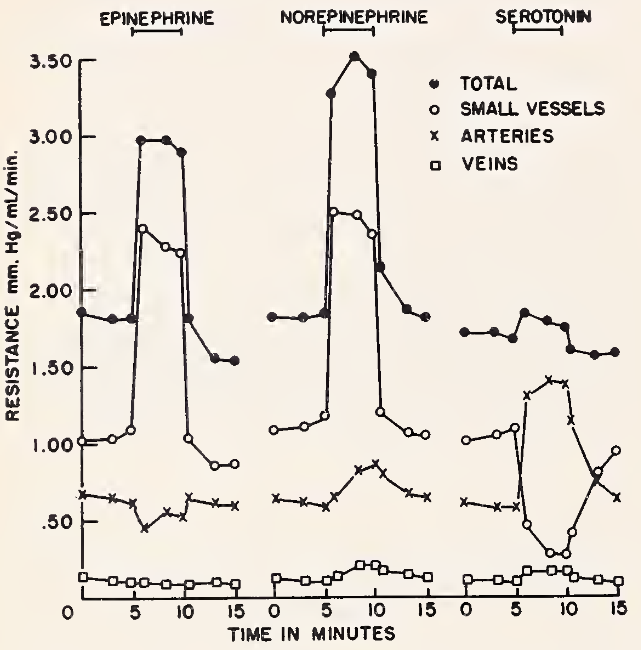

Fig. 8. Average effect of epinephrine, norepinephrine and serotonin upon total and segmental vascular resistances in eleven dog forelegs. Foreleg blood flow rates maintained constant at average values of 69, 69 and 75 ml./min., respectively. Epinephrine hydrochloride, 1-norepinephrine bitartrate monohydrate and serotonin creatinine sulfate infused into brachial artery at rates of 5, 5 and 10 γ/min., respectively. Norepinephrine changes illustrate homogeneous active constriction, epinephrine active small vessel constriction concomitant with passive artery dilatation and serotonin active artery and vein constriction concomitant with active small vessel dilatation.

Combinations of Active and Passive Resistance Changes.—The epinephrine response with blood flow rate maintained constant illustrates active small vessel constriction concomitant with passive artery dilatation (Fig. 8). The artery segment dilated because its average intraluminal pressure increased 75 mm. Hg. The dilatation is less, however, than would be expected from the pressure change alone[2] indicating that epinephrine also tends to constrict arteries.[5] A similar combination of directionally opposite active and passive segmental changes occurs following venous obstruction.[2] This maneuver, when carried out with blood flow rate constant, is usually associated with active small vessel constriction and passive artery and vein dilatation (Fig. 9). Since pressures increase in all vessels, the small vessel constriction is active in nature whereas the dilatation in arteries and veins is passive. The small vessel constrictor response can be converted to a dilator response by destroying nerve function in the limb. The response has been called the 'veni-vasomotor' reflex by Burton's group[6-10] and the 'venous-arteriolar' reflex by Haddy and Gilbert.[2,11]

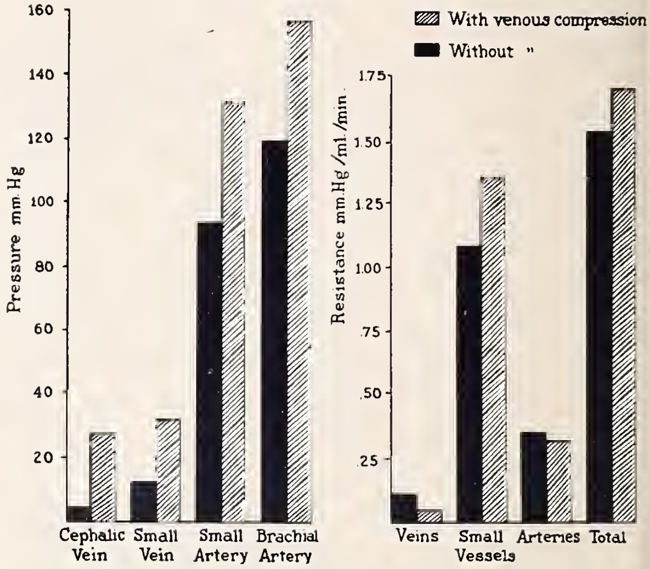

Fig. 9. Effect of venous obstruction upon vessel pressures and segmental vascular resistances in foreleg of pentobarbitalized dog. Blood flow rate maintained constant at 75 ml./min. Venous obstruction accomplished by tightening a tourniquet high on the leg. Response illustrates passive vein and artery dilatation concomitant with active small vessel constriction.

Heterogeneous Active Resistance Changes.—Directionally opposite active changes in various segments were an unexpected finding. During cold exposure, all segments usually constrict.[12] Upon return to room temperature, the small vessel segment immediately begins to dilate. However, this is not always true of the vein and artery segments. In some instances the veins continue to actively constrict during the rewarming period at a time when small vessels actively dilate. Kelly and Visscher[13] have reported changes which likely represent the same event following the release from faradic sympathetic nerve stimulation. The cold-induced venous constrictions are associated with high levels of small vein pressure. Using these catheterization techniques in humans, Stead and Wallace[14] have reported small vein pressures as high as 40 to 50 mm. Hg. during cold exposure. Active artery constriction also occasionally occurs during the rewarming period concomitant with active small vessel dilatation (Fig. 10). Active artery dilatation concomitant with small vessel constriction is observed in nerve intact forelegs if CO_2 tension and H^+ concentration are decreased by hyperventilation.[15] Since the two responses are directionally opposite and of approximately equal magnitude, total resistance does not change significantly. On the other hand, the opposite changes are observed when H^+ concentration and CO_2 tension are increased by ventilation with 20 per cent CO_2.

The most striking example of directionally opposite active changes in segmental resistances yet observed is that associated with local serotonin administration.[5] Figure 11 shows that intra-arterial injection of this substance results in little change in flow rate and large artery pressure but a large decrease in small artery pressure associated with an increase in small vein pressure. Large vein pressure does not change. Resistance calculations (Fig. 8) show a striking increase in artery resistance and a decrease in small vessel resistance. Venous resistance also increases and in some instances is associated with small vein pressure levels as high as 35 mm. Hg. The over-all result is little change in total resistance. An examination of the direction of pressure changes in the various segments indicates that all the caliber changes are active in nature. These findings may explain how serotonin produces peculiar skin color changes without predictable blood pressure changes when injected into intact animals or man.

Numerous investigators have noted that administration of serotonin produces unpredictable changes in systemic arterial blood pressure. Page and McCubbin[16] observed that the direction of change seemed to be related to the pre-existing level of resistance in the system. The present studies[17] offer an explanation for their finding. In the experiment illustrated in Figure 12, administration of serotonin decreased total resistance when nerves were intact and control total resistance high. Following nerve section, control total resistance decreased because of small vessel dilatation. In the latter low resistance preparation, administration of serotonin increased total resistance because the small vessels, being maximally dilated already, could not further dilate in order to offset constriction in arteries and veins. In a series of experiments recently completed, four levels of small vessel resistance were studied in the same animal. The magnitude of serotonin-induced small vessel dilatation was directly related to the pre-existing level of small vessel resistance.

Local Anesthesia

Experiments in trained unanesthetized animals suggest that a number of the above events may occur spontaneously in normal animals or may be precipitated by certain pathological conditions. Average, small and large vein pressures in unanesthetized animals are not greatly different from those under general anesthesia.[1] The respiratory waves are not so readily apparent since the ventilatory cycle is more rapid and less regular. However, if small vein pressure is observed over a long time period in wakened animals, it sometimes spontaneously fluctuates over wide ranges in the absence of significant changes in large vein pressure.[1]

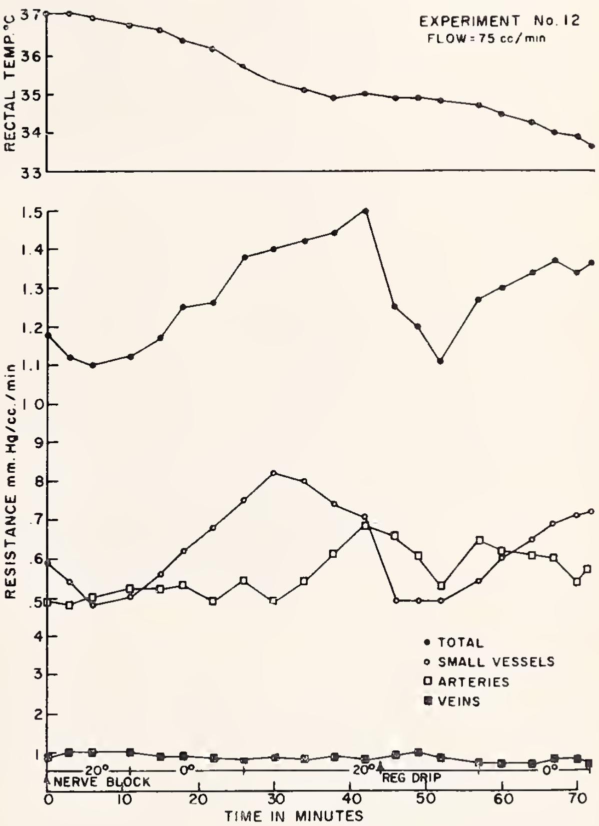

Fig. 10. Unusual effect of cold exposure upon total and segmental vascular resistances in foreleg of pentobarbitalized dog. Foreleg blood flow rate maintained constant at 75 ml./min. Entire transverse diameter of foreleg blocked with 2 per cent procaine at level of elbow at point indicated. O° C exposures accomplished by placing animal in cold room for periods indicated. Phentolamine methanesulfonate infused into brachial artery at rate of 600 γ/min. from forty-third to seventy-second minute. Experiment illustrates active artery constriction concomitant with active small vessel dilatation resulting in continued rise in total resistance during first rewarming period.

Studies have also been made in seventeen unanesthetized dogs with artificially-produced pulmonary stenosis and tricuspid insufficiency.[3,18] Eight were compensated and nine were decompensated at the time of study. The independent variability of small vein pressure was even more apparent in these animals than in those with normal circulatory systems. The gradient from small to large vein, integrated over long time periods, was 11.3 mm. Hg. and is to be compared to a value of 6.0 mm. Hg. from a group of normal wakened animals. Figure 13 shows that exercise also elevates the gradient from small to large vein in normal animals.[1] Elevated venous gradients were also observed in four edematous extremities. In one case, the measurements were made in a wakened dog with edema due to cardiac decompensation produced by artificially-induced mitral stenosis.[3] The three others were under general anesthesia and the hindleg edema was the result of chronic iliac and femoral arteriovenous fistulae.[1]

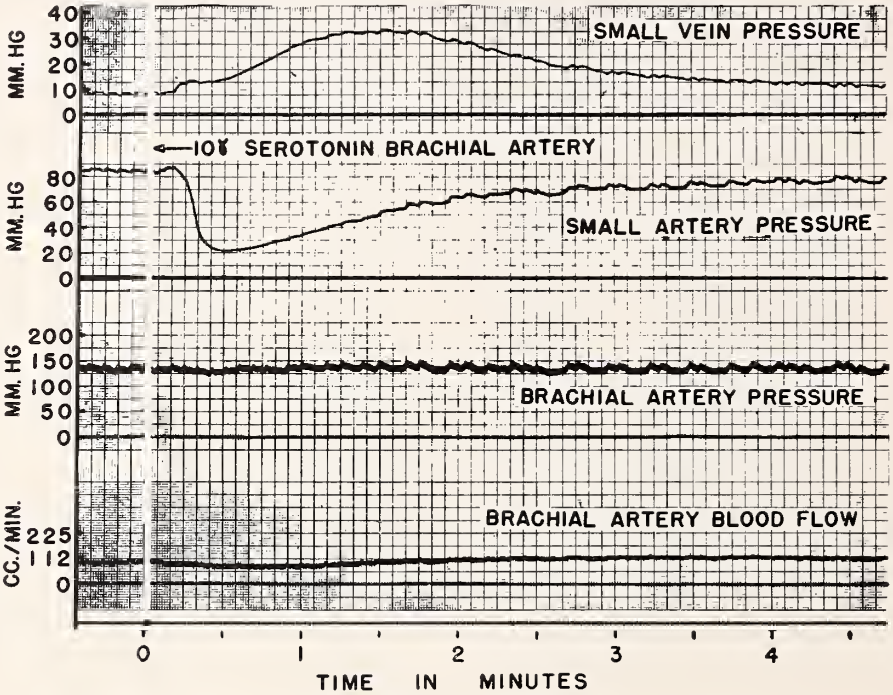

Fig. 11. Effect of serotonin creatinine sulfate upon vascular pressures and blood flow rate in foreleg of pentobarbitalized dog. Instantaneous injection made just distal to branchial artery flowmeter. Cephalic vein pressure did not change significantly.

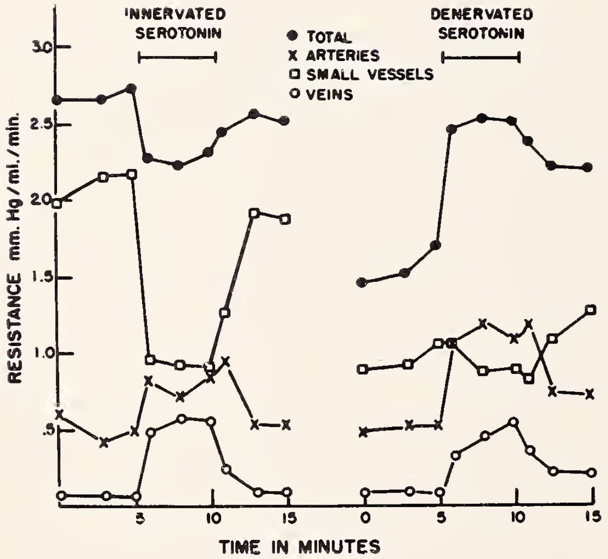

Fig. 12. Effect of nerve section upon total and segmental vascular resistance responses to serotonin in foreleg of pentobarbitalized dog. Blood flow rate maintained constant at 50 ml./minute. Serotonin creatinine sulfate infused into brachial artery just distal to blood pump at rate of 10 γ/min. Experiments executed immediately before and after cutting all nerves high in the foreleg. Experiment illustrates the mechanism whereby serotonin induced total resistance decrease in a high resistance foreleg is converted to a total resistance increase in a low resistance foreleg.

Gradient changes may arise from variation in blood flow rate or resistance. Since the animals were awake, flow rates are not available. However, large changes in flow rate produce minimal variation of small and large vein pressures in anesthetized animals (Fig. 3).[2] This suggests that the above-mentioned pressure and gradient changes in the venous segment of wakened animals are truly due to active venomotion.

A study of the venous system of humans is in progress. Simultaneous measurements have been made of pressures in the antecubital vein and a subcutaneous small vein at the base of the fingers in the same extremity. The catheter is introduced into both veins through needles inserted through the intact skin. In ten normal subjects, the values for small and large vein pressure average 11.6 and 8.0 mm. Hg., respectively. To date, the large spontaneous fluctuations of small vein pressure mentioned above have not been observed in humans. It should be noted, however, that it has not always been possible to advance the catheter as far distally in the human as in the dog.

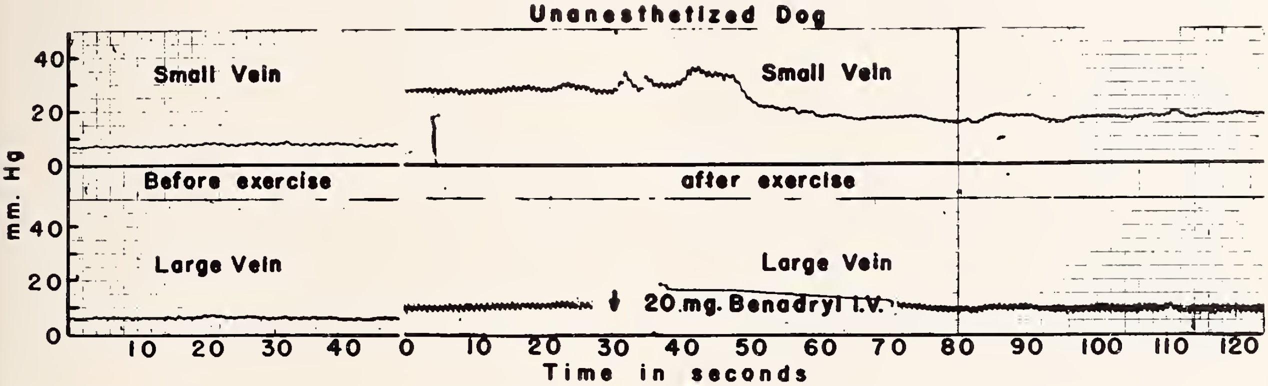

Fig. 13. Effect of exercise upon small and cephalic vein pressures in foreleg of trained wakened dog. Catheters inserted under local procaine anesthesia. Animal lying on side during period pressures were recorded. Recordings immediately before and after animal was made to run 150 yards at the approximate rate of 5 miles/hr. Catheters were disconnected from strain gauges, filled with a saline-heparin solution, tightly stoppered and taped to animal's leg without disturbing their positions in the veins for the period of exercise. Drug injection into cephalic vein catheter during period indicated.

Discussion

These observations indicate that macroscopic arteries and veins may contribute significantly to the maintenance and adjustability of resistance to blood flow in dog limbs. Further, they suggest that arteries, small vessels, and veins are independent resistances. Their magnitudes may actively or passively change in the same or opposite direction.

These statements are perhaps best illustrated by serotonin administration. Serotonin appears to shift the major portion of total resistance from small vessels to arteries and veins. The segmental changes are large and of opposite direction.

Bore changes in various segments are important for two reasons. They vary volume distribution of blood within these segments. Changes in venous volume are especially important because they directly influence the rate of cardiac filling and thereby, the cardiac output. Bore changes also result in pressure variations which, in turn, may influence the transport of water and electrolytes across capillary membranes. Active venous constriction was associated with small vein pressures as high as 50 mm. Hg. in some instances. Since capillary pressures in open beds cannot be lower than small vein pressure, net transport of fluid was likely out of the capillary in these instances. Therefore, further knowledge about venous resistance and small vein pressure changes may shed light upon the mechanism of edema production and tissue water storage, in general. Changes in artery caliber likely do not influence blood volume distribution as acutely as do variations in venous caliber. They do, however, greatly influence capillary pressure and the direction of change is the same as that caused by changes in arteriolar caliber.

These studies emphasize another rather practical point. Since veins can actively constrict and dilate, there need be no fixed correlation between small vein pressures and those in larger, more central veins. Reliance upon large vein pressure changes to indicate what is going on at the periphery is, therefore, obviously improper. The same applies to the arterial side of the circuit.

Conclusions

Methods are described for measurement of resistance in arteries, small vessels and veins of the dog foreleg. Observations under various circumstances indicate that these vessel segments constitute independent resistances whose magnitudes may vary in the same or opposite directions through both active and passive mechanisms. Veins and arteries of greater than microscopic size may, under certain conditions, contribute greatly to the maintenance and adjustability of resistance to blood flow across systemic vascular beds.

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DIET, PHYSICAL ACTIVITY AND THE SERUM CHOLESTEROL CONCENTRATION

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Experimental Cerebrovascular Disease

A Review

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RELATIVELY few studies of experimental cerebrovascular disease in animals have been recorded. These studies have tended to lag behind clinical and pathologico-anatomic investigations and too frequently have served as confirmatory work after clinical impressions have been virtually accepted.

It is obvious that investigation with laboratory animals cannot be related directly to human disease. No animal has a cerebral vascular supply entirely comparable to that of man, and most, if not all, animals have a considerably greater cerebral circulatory reserve. For example, my associates and I[1] have learned that 50 per cent of dogs which have had both vertebral arteries, both common carotid arteries and all their terminal branches ligated in the neck simultaneously will survive and recover near normal function. Another point regarding the limitations of these studies is that young animals ordinarily are used; seldom, if ever, do these animals have diseased cerebral blood vessels. It is possible to use dogs in which atherosclerosis has been produced by thiouracil and high-cholesterol diets. However, even when rather pronounced changes have occurred in many arteries of the dog's body, the change in the cerebral vessels has been little or none.[2]

Production of Experimental Cerebral Infarction

My colleagues and I have been interested in satisfactory methods for the production of infarcts in order that we might determine how these infarcts can be altered by various procedures or drugs. Ligation of individual arteries in the neck rarely causes focal infarction in animals. Craniotomy followed by occlusion of individual cerebral arteries has been performed in a number of studies. In a high percentage of instances, occlusion of the middle cerebral artery with a clip in dogs or monkeys has produced hemispheric infarction, but with considerable variation in size, which is one of the difficulties in interpretation of data, regardless of the method used for producing the infarcts.

A number of different foreign substances, including various seeds, sand, air, iron filings and stainless-steel ball bearings, have been injected into the arteries of the neck in animals. These methods have met with varying success in producing lateralized focal infarction of any predictable nature. Denny-Brown and associates[3] produced hemorrhagic cerebral infarction by abruptly distending a cerebral vein with an isotonic solution of sodium chloride, the infarct occurring in the area drained by that vein.

Effects of Other Factors on Experimental Cerebral Infarcts

Rosomoff and Holaday[4] noted that hypothermia in dogs reduced the cerebral blood flow an average of 6.7 per cent of the control flow per degree when the temperature was reduced from 35° to 25° C. Cerebral consumption of oxygen was reduced correspondingly. Rosomoff[5] found that dogs in which clips were placed to occlude the middle cerebral artery had much smaller infarcts and many fewer clinical signs when they were under hypothermia than did control normothermic dogs.

Hypotension induced by drugs or by bleeding has been found in several studies to affect adversely the size of cerebral infarcts in cats, dogs and monkeys after a clip has been placed to occlude the middle cerebral artery.

Meyer and co-workers[6] measured the availability of oxygen in various zones of cerebral infarcts in cats and monkeys by the use of an electropolarograph. They noted in unstable parts of the infarcts that the available oxygen passively fol-

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lowed decreases in the blood pressure, and they concluded that maintenance of blood pressure at optimal levels was the most important factor in maintaining the available anastomotic circulation. They also noted that the pattern and speed of recovery of the infarcts were not affected by cervical sympathectomy, stellate ganglionectomy, sectioning the seventh cranial nerve and breathing 5 per cent carbon dioxide and 95 per cent oxygen.

Corday and associates[7] reported more pronounced electroencephalographic changes in the homolateral hemisphere when monkeys with one internal carotid artery partially or completely occluded were subjected to hypotension; these changes were reversed readily when the blood pressure was restored to normal.

Hypertension induced by drugs after the production of cerebral infarcts in dogs increases the extent of hemorrhagic infarction.

Production of Hemorrhagic Infarction

Hain and co-workers[8] concluded that they could produce hemorrhagic infarcts in dogs by occluding the middle cerebral artery proximal to the origin of the perforating branches. They obtained pale infarcts when the occlusion was distal to these branches. It was thought that collateral circulation from these branches was the origin of the hemorrhagic character of the infarcts and that the blood was present by virtue of diapedesis before necrosis of the blood vessels occurred.

Globus and associates[9] produced infarcts by occluding the middle cerebral artery in dogs. After varying periods of delay, phenylephrine (neosynephrine) hydrochloride was given intravenously on several occasions, thus producing fresh focal hemorrhages in two-thirds of the dogs. Although hemorrhagic infarction and frank hemorrhage were not clearly differentiated in their study, the photographs revealed that frank hemorrhage occurred on several occasions, and intraventricular hemorrhage was present in one dog in which the infarct was thirty days old.

Fazio and Sacchi[10] induced infarcts in dogs by air emboli and thought that hemorrhagic infarction was increased by the delayed intravenous administration of epinephrine (adrenalin). They concluded that infarction was increased further by venous stasis produced by a head-down position plus compression of the jugular veins.

Harvey and Rasmussen[11] produced hemorrhagic cerebral infarcts in monkeys by complete occlusion of the middle cerebral artery for fifty minutes. This, in effect, is re-establishment of the circulation through damaged tissue. Welch,[12] in his classic study of hemorrhagic infarction of the small intestine of the dog in 1887, produced severely hemorrhagic infarction when all collateral channels had been previously excluded, and he then incompletely occluded, for a prolonged period, the superior mesenteric artery so that the blood barely flowed. I am not aware that a similar study has been performed in regard to the cerebral circulation.

Other Experimental Cerebrovascular Diseases

Hypertensive encephalopathy was produced in rats by Byrom.[13] The hypertension was effected by excising one kidney and constricting the opposite renal artery. The encephalopathic symptoms were relieved promptly by abolition of the hypertension. Organic lesions were found in 55 per cent of the rats at necropsy; these consisted of focal arterial necrosis, small infarcts and arterial or capillary hemorrhages. Diffuse spasm in small arteries and diffuse and focal spasm in larger arteries were noted in these rats by observing the brain through a cranial window. Byrom concluded that the encephalopathy and arterial necrosis both resulted from arterial spasm.

Experimental cerebral arterial aneurysms have received little attention. McCune and associates[14] produced aortic aneurysms in dogs by injecting nitrogen mustard under the adventitia. Similar procedures have been directed toward the carotid artery in the neck, but aneurysms have not been consistently produced in the vessels of the neck or the intracranial arteries.

Cerebral Infarction and Anticoagulant Therapy

My associates and I have found two useful methods for the production of cerebral infarction. The first method[15] involves the introduction of 0.2 to 0.3 ml. of liquid vinyl acetate into one internal carotid artery of dogs; this material promptly hardens on contact with the blood and occludes the internal carotid artery, usually the middle cerebral artery and sometimes other cerebral arteries. The material usually is in a continuous strand within the arterial tree. The other method[16,17] involves similar injection of approximately the same amount of a forty-eight-hour-old homologous blood clot. The obstruction in this technique should be thought of as a shower of emboli consisting of fragments of clot that lodge in positions such as the trifurcation of the middle cerebral artery. The age of the clot (forty-eight hours) is an important consideration in this technique.

TABLE I. EXTENT OF HEMORRHAGE IN CEREBRAL INFARCTS PRODUCED BY VINYL ACETATE AND BY CLOTS

Hemorrhage, Per Cent	Vinyl Acetate		Homologous Clot	
	Number	Per Cent	Number	Per Cent
0-30	13	93	2	11
31-60	0	0	4	22
61-90	0	0	4	22
91-100	1	7	8	45

Both these methods have yielded lateralized cerebral infarcts in approximately 75 per cent of the animals. This figure represents those infarcts large enough to be readily detected clinically, which means that the animal has hemiparesis or forced circling or both. Of the dogs so affected, about half fail to survive the first two days after the infarct is produced, which limits the value somewhat. The other half of this group survive indefinitely and recover to varying degrees.

One of the difficult problems in such studies is to convey to other persons the extent of an infarct and its hemorrhagic nature without detailed description. Because of this, we have made a practice of cutting each brain into seven standard coronal sections, and a scale drawing on a printed form has been made, representing structures in each of these standard sections. The infarcts then are outlined on the printed sheets, the hemorrhagic zones being noted in red and the nonhemorrhagic parts in blue. These areas then can be traced with a planimeter, which gives the total area of infarction in the seven sections. Thus, the complexities of an infarct can be reduced to numbers, which can be used for comparison. It is not possible to indicate satisfactorily the density of these areas. The limitations are obvious, but the technique has proved useful.

Practically all the infarcts produced by vinyl acetate were less than 30 per cent hemorrhagic (Table I). The one extremely hemorrhagic lesion was an exception to the relatively unaltered infarcts produced by this method. An earlier group of seventeen infarcts produced by vinyl acetate further support this experience. On the other hand, the infarcts produced by injection of clots varied in the amount of hemorrhage, but two-thirds were more than 60 per cent hemorrhagic (Table I).

Alteration of Infarcts by Anticoagulants.—Since anticoagulants have had such increasing clinical use in the last few years in cerebrovascular diseases, my colleagues and I have conducted several studies aimed toward increasing our knowledge about alterations of experimental cerebral infarcts by these agents. We were interested in whether cerebral infarcts had a more hemorrhagic character when anticoagulants were given immediately after the infarct was produced. Since many infarcts are strikingly hemorrhagic without anticoagulants and since such great variability occurs in this feature, one must depend on the over-all picture in a number of animals to obtain any useful data.

TABLE II. EFFECT OF ANTICOAGULANTS ON EXTENT OF HEMORRHAGE IN CEREBRAL INFARCTS PRODUCED BY VINYL ACETATE

Hemorrhage, Per Cent	Control Dogs		Dogs Given Anticoagulants	
	Number	Per Cent	Number	Per Cent
0-30	13	93	16	64
31-60	0	0	8	32
61-90	0	0	1	4
91-100	1	7	0	0

When ethyl biscoumacetate (tromexan) and bishydroxycoumarin (dicumarol) are given, it requires a little more than twenty-four hours to reduce the prothrombin activity in dogs to the therapeutic range. Our initial experience[18] involved thirteen animals which were given anticoagulants on the same day the infarct was produced and survived at least thirty hours, allowing the anticoagulants to become effective. These infarcts were produced with vinyl acetate, which, as already noted, usually produces relatively nonhemorrhagic lesions. Eleven of these infarcts were strikingly hemorrhagic when compared to the lesions in thirteen control dogs which also survived at least thirty hours but did not receive anticoagulants. A considerable range of hemorrhagic infarction often was noted even within the same infarct. In four animals, the entire infarct was occupied by hemorrhage, and the same condition plus intraventricular hemorrhage was noted in a fifth dog. The prothrombin activity in this group of animals was made excessively low intentionally, the average of the lowest values for each animal being 5 per cent of the normal prothrombin activity.

We[19] then conducted a similar study, taking special care to maintain the prothrombin activity at approximately 15 to 30 per cent of normal. This study was limited to dogs which survived at least forty-eight hours, so that full anticoagulant activity could be effected. The hemorrhagic zones were mapped out in the standard sections for comparison with a group of fourteen control animals (Table II). It is apparent that the infarcts in the group which received anticoagulants were significantly more hemorrhagic; however, one infarct in the control group was almost entirely hemorrhagic, and two dogs which received anticoagulants had no evidence of hemorrhage in their infarcts.

TABLE III. EFFECT OF ANTICOAGULANTS ON SIZE OF INFARCTS PRODUCED BY CLOTS

Infarct, Per Cent	Control Dogs	Dogs Given Anticoagulants
0-3	5	8
4-7	3	5
8-16	5	1
16+	1	0

All control animals which lived for three days after an infarct was produced survived indefinitely. On the other hand, of the group which received anticoagulants and survived at least three days, eight of twenty (40 per cent) died before the termination of the study on the twelfth day. One must conclude, then, that anticoagulants in this study increased the hemorrhagic character of the infarcts and also increased the hazard to the animals. It should be emphasized that this applies to fresh hemispheric infarcts that are completed.

In another group of dogs, the infarcts were produced by forty-eight-hour homologous blood clots.[20] These animals had received anticoagulants (tromexan and dicumarol) for two days prior to the day on which the infarcts were produced, so that therapeutic reduction of prothrombin activity existed at that time. The prothrombin activity was maintained close to the therapeutic range. The problem of hemostasis was increased at the time of the operative procedure for the injection, but it was by no means insurmountable.

Since this method of production of infarcts constitutes a shower of clot fragments, or emboli, we tried to estimate whether there was any protection afforded the dogs which were given anticoagulants prior to injection of the clots. This was done by comparing the total area of infarction in the seven standard sections with the findings in a control group. This comparison was limited to those infarcts more than twenty-four hours old, so that they would be well demarcated (Table III). A slight difference was noted in favor of the dogs which received anticoagulants, but it was not striking. Likewise, the over-all percentage of animals which had infarcts in this group was not significantly different from that usually obtained by injection of clots. Despite a therapeutically reduced prothrombin activity at the time of the injection of the clot, fragments of clot were found grossly in the major arteries at or near the base of the brain in almost half of the animals.

TABLE IV. EFFECT OF ANTICOAGULANTS ON EXTENT OF HEMORRHAGE IN CEREBRAL INFARCTS PRODUCED BY CLOTS

Hemorrhage, Per Cent	Control Dogs		Dogs Given Anticoagulants	
	Number	Per Cent	Number	Per Cent
0-30	2	11	2	10
31-60	4	22	2	10
61-90	4	22	4	20
91-100	8	44	12	60

As noted previously, infarcts produced by injection of homologous clots are relatively hemorrhagic lesions even in the absence of influence from other agents. We have compared a group of controls of this type with a group which received anticoagulants (Table IV). A wide range in the extent of hemorrhage occurred in each group, and the anticoagulants apparently had little effect on the cerebral infarcts when compared with control infarcts that ordinarily have a hemorrhagic character. The relatively small differences indicated in the incidence of infarcts with maximal hemorrhage are not statistically significant. However, the slight difference noted in Table IV is increased by the knowledge that frank hemorrhage was present within two of the infarcts in the dogs receiving anticoagulants, in one of which intraventricular hemorrhage was noted. This was not seen in any of the infarcts of the control animals. Including all our studies, hemorrhage has not been seen in the brain of an animal which received anticoagulants, unless it was within an infarct or had ruptured into the ventricles.

Comment

It should be emphasized that these studies are not an attempted indictment of the clinical use of anticoagulants in cerebrovascular disease. It is noteworthy that, whenever an excessively hemorrhagic infarct was present, it occurred in recently produced, completed infarcts in one hemisphere. There is little evidence, clinical or experimental, that anticoagulants are beneficial in this particular category of cerebrovascular disease. It is hoped that the results of studies such as these will be used as a "caution sign," so that the indications for and limitations of anticoagulant therapy will be clearly distinguished.

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Diseases of Small Arteries and Arterioles

Emphasizing Fibrinoid and Hyaline Changes

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THE FIBRINOID CHANGE represents a morphologic alteration involving small arteries and arterioles which occurs in several major vascular diseases. These diseases include such varied entities as malignant or accelerated hypertension, eclamptic toxemia of pregnancy, diabetes mellitus, the collagen diseases especially when associated with an arteritic component, thrombotic thrombocytopenia and symmetrical renal cortical necrosis. Moreover, certain experimentally induced states also express themselves morphologically by the presence of "fibrinoid" within small arteries and arterioles. Notable among these are the experimentally induced hypertensive cardiovascular states and the disturbances associated with the generalized Shwartzman reaction.

It follows that "fibrinoid," as encountered in small arteries and arterioles, represents a fundamental reaction which must be reckoned with in any consideration of the pathogenesis of the various disorders in which it is encountered.

Observers interested in the above listed group of diseases have concerned themselves with the characteristics of fibrinoid and have, from time to time, considered the complex problem of the genesis, the meaning and the disposition of this material.

Altshuler and Angevine[1] made extensive studies dealing with the problem of fibrinoid and added to the appreciation of this material. According to these workers, a common feature of fibrinoid formation is the precipitation of acid mucopolysaccharide from the ground substance of connective tissue. This disturbance was considered to give rise to the formation of a homogeneous, eosinophilic, relatively acellular substance with some properties of fibrin as described by Neumann. In these studies, distinction between fibrinoid as encountered in connective tissues and that within blood vessels was not stressed.

More recently, interest in fibrinoid has received major impetus by observations dealing with this material as observed in association with the Shwartzman phenomenon and in the collagen diseases by Thomas, Good, Smith, Brunson and co-workers.[2-8] These workers have stressed the possibility of a hematogenous source of fibrinoid in these conditions.

Brunson and co-workers clearly described the disposition of fibrinoid in the Shwartzman reaction and depicted certain of its histologic properties. The possibility that this material may be derived from fibrinogen or altered fibrinogen was entertained. Potentiation of the "fibrinoid deposits" by acidic polymers was demonstrated while heparin appeared to play a protective role. In essence, the hypothesis considered by these workers placed the fibrinoid of the Shwartzman reaction in the role of an infiltrative or embolic material derived from altered plasma proteins.

The diseases which are attended by the vascular and connective tissue fibrinoid changes may be associated with the development of proteins in the plasma which are precipitated in the cold by heparin.[8] These proteins have certain characteristics of fibrinogen.

The use of the fluorescent antibody technique of Coons has indicated the presence of fibrin within the fibrinoid of connective tissues as well as within the fibrinoid of blood vessels.[9] Whether the fibrin present results from the permeation by plasma proteins (including fibrinogen and its product fibrin) of a non-hematogenous substance ("fibrinoid"?) previously developed within the blood vessels or whether it represents a primary disturbance remains to be demonstrated.

This brief consideration may be taken to indicate a widespread occurrence of the material known as fibrinoid in a series of vascular diseases

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TABLE I. MAJOR STAINING AND HISTOCHEMICAL FEATURES OF FIBRINOID

Material	H and E	Mallory Aniline Blue Masson Trichrome	PAS	Lipids Oil Red O Sudan Black B Nile Blue Sulfate Schultz	Free Carbonyl	Protein SH	K	Congo Red	Xantho-proteic	Millon	Indole	CA
Vascular Fibrinoid	Eos.	Red	Purple (+)	+	+	+	+	+	—	—	—	—
Connective Tissue Fibrinoid	Eos.	Red	Purple (+)	—	—	—	—	—	+	+	+	+
Fibrin	Eos.	Red	Purple (+)	—	—	Weak +	Weak +	Weak +	+	+	+	+

which likely are initiated by different primary causes. Approaching the problem from this view, it is plausible to expect a common means for the development of this morphologic expression of injury in these various states. The two concepts mentioned above have attempted to explain this common path of expression by invoking, on the one hand, changes in the ground substance of connective tissue and, on the other, the deposition of materials having a hematogenous source.

Our interest in the problem of the fibrinoid changes in small arteries and arterioles began with observations of the hypertensive cardiovascular state following renal ablation of the dog.[11] Subsequently, observations were extended to include tissue changes in malignant and benign hypertension of man, in kidneys from patients having suffered from diabetes, renal cortical necrosis, thrombotic thrombocytopenia and in the kidneys from rabbits following the Shwartzman reaction. These studies have led us to consider an approach to the genesis of vascular fibrinoid which differs fundamentally from the two approaches mentioned above. The thesis which we support considers that the vascular fibrinoid of these various states is derived mainly from disintegrated vascular smooth muscle. The latter may be termed the concept of the *autochthonous origin of vascular fibrinoid.* Features supporting this concept are considered in this discussion.

Fibrinoid: Connective Tissue versus Vascular Types

Fibrinoid has been described within blood vessels in the conditions listed above and within connective tissues in certain disorders, that is, in rheumatic fever, rheumatoid arthritis, disseminated lupus erythematosus, as well as in the base of peptic ulcers, between the ends of poor-healing fractures and within the placenta.

In the course of considering fibrinoid it has appeared of advantage to us to distinguish between the "connective tissue" and "vascular" types. This distinction has been made possible, primarily, because of markedly divergent staining and histochemical characteristics.[10]

Major staining and histochemical features of fibrinoid are given in Table I. With the procedures used it is evident that connective tissue fibrinoid, as observed in certain conditions, gives certain staining and histochemical reactions similar to those of fibrin. Both materials were eosinophilic, intensely red with Mallory's aniline blue and Masson's trichrome stains and positive (purple) with the periodic acid-Schiff reagent (PAS) procedure. Both ingredients failed to yield evidence for the presence of various lipids and free carbonyl groups. Fibrin gave weak reactions for protein-bound SH, free potassium and acid esters of polysaccharide origin (using Congo red) while connective tissue fibrinoid was negative for these procedures. Both fibrin and connective tissue fibrinoid gave distinctly positive reactions with the xanthoproteic, Millon, indole and calcium reactions.

The histologic pattern for vascular fibrinoid has been consistent. The material was eosinophilic, red with Mallory's aniline blue and Masson's trichrome stains and purple (positive) with the PAS procedure. With these approaches, vascular fibrinoid, connective tissue fibrinoid and fibrin were similar. Vascular fibrinoid, however, yielded intensively positive reactions with various lipid-demonstrating procedures and with the procedures for free carbonyl groups, for protein-bound SH and for free potassium. The Congo red procedure was positive when frozen sections were used. On the other hand, xanthoproteic, Millon, indole and calcium reactions were negative.

In summary, with certain procedures vascular and connective tissue fibrinoid are similar in appearance. With other procedures these two materials give reactions which mirror each other (staining patterns are reversed) as depicted in Table I. The characteristics of vascular fibrinoid emphasize an eosinophilic appearance, a positive reaction for free aldehyde groups of polysaccharide origin (PAS) and acid esters of polysaccharide origin (Congo red), positive reactions for various lipids and fats (oil red O, Sudan black B, Nile blue sulphate, Schultz reaction), positive reactions for free carbonyl groups, protein-bound sulf-hydryl groups and free potassium. The term "vascular fibrinoid" as considered in this discussion, is thus reserved for material encountered in various vascular disorders which stress these characteristics.

Fibrinoid and Vascular Diseases

The appearance and distribution of "vascular fibrinoid," as identified by the characteristics listed above, in various vascular diseases and experimental states may be utilized in support of a concept considering its origin.

1. *Renal Ablation of Dog.*[11-18]—Bilateral nephrectomy of the dog under certain conditions is attended by systemic arterial hypertension and the development of cardiovascular lesions. The vascular lesions involve especially the small arteries and arterioles which constitute the intrinsic vasculature of the viscera. The appearance of the arterial and arteriolar lesions, in turn, is mostly dependent on the management given this experimental preparation.

A high intake of sodium[19] or a lesser intake of sodium plus a protein-containing diet[20] is associated with a rapid rise in the arterial pressure and widespread disruption of small arteries and arterioles. Prolongation of life by electrolyte balance, diet and dialysis alters appreciably the characteristics of the arterial-arteriolar lesions. In both the acute and the subacute-chronic phases of this preparation "vascular fibrinoid" is encountered. By arresting the course of this experimental preparation at various times, a sequential relationship can be demonstrated for the various expressions of the vascular disease.

Fulminant Acute Phase.—A rapid disruption of arteries and arterioles in the renoprival state is attended by the development of "fibrinoid" within the walls of these vessels. The relationship of this material to the vessel wall and its distribution are considered pertinent to the concept of its origin.

The fibrinoid is found overwhelmingly within the area of the media where smooth muscle normally exists. The appearance of the fibrinoid describes an inverse relation to the presence of smooth muscle. Where only a segment of the circumference is involved, this segment is in direct continuity with the normal appearing media. More important, however, are transitional changes between the normal muscle and the fibrinoid. In the transitional zone, swollen muscle fibers with pyknotic nuclei may be seen. Non-nucleated masses with the configuration of swollen muscle fibers may be seen and the substance of these masses has the characteristics of "vascular fibrinoid." These individually swollen masses, which are considered to represent damaged smooth muscle fibers, mesh imperceptibly with the typical fibrinoid material. It is possible to encounter the area of the inner half of the media showing the fibrinoid change while the outer half has intact smooth muscle. Centripetal spread of the fibrinoid into the lumen by contraction of the rim of smooth muscle near the adventitia appears plausible.

The fibrinoid in the media appears to have properties of a hydrogel. At any rate, it may spread into the adventitia in a pseudopod fashion or it may be continuous with similar material within the lumen. In the lumen the configuration may be that of a polypoid mass or it may fill the lumen. In areas where the fibrinoid appears smeared and spread out, intact erythrocytes may be seen within its substance. The endothelium may or may not be disrupted but classical thrombosis is not observed. Capillaries which are obstructed and distended with fibrinoid may be encountered in the vicinity of arteries and arterioles displaying the fibrinoid change.

Subacute and Chronic Phase.—Dogs which are sustained by dialysis for ten to over 100 days following renal ablation not only maintain the hypertensive state but the characteristics of the arterial and arteriolar lesions are altered. Within ten to thirty days the fibrinoid changes, as described above, become less prominent. The material in the media begins to assume the appearance of hyalin. Occasionally nuclear remnants are seen within the hyalinizing substance. Later there is distinct hyaline thickening of arterioles. Along with the hyaline changes, connective elements are observed in the intima, media or adventitia. Such vessels display narrowed lumina and thickened walls. The characteristics are those of arteriolarsclerosis.

The hyalin of the hyaline arteriolarsclerosis following renal ablation displays the staining and histochemical characteristics of "vascular fibrinoid" although by ordinary stains it appears compact and smoother than fibrinoid. Just as transitional changes are observed between the smooth muscle of the media and vascular fibrinoid, so transitions may be encountered between vascular fibrinoid and vascular hyalin.

2. *Essential Hypertension in Man.*[11,15-17]—In the accelerated form or malignant hypertension of man "vascular fibrinoid" is encountered. Although the material may be observed in the arterioles of various viscera, such as pancreas and periadrenal area, it is most prominent within the kidney. The characteristics and configuration of this material are identical to those encountered in the dog following renal ablation.

Of particular interest is the encounter of elongated or globular masses of "fibrinoid" of the vascular type within the glomeruli in malignant hypertension. In isolated examples it is possible to encounter fibrinoid in the wall of the afferent arterioles, within their lumina and from here in direct continuity with the same material in glomerular capillaries. This association led to the suggestion that the glomerular fibrinoid was derived from the arteriolar fibrinoid by embolization. For the same reasons given in the section on renal ablation the origin of the arteriolar fibrinoid of malignant hypertension from vascular smooth muscle has been proposed. This is in keeping with the view of workers who have designated this lesion as arteriolarnecrosis.[21]

In benign hypertension, the arteriolar hyalin appears identical to that described above following renal ablation plus prolonged survival. In a study destined to compare the characteristics of the hyalin of benign arteriolarsclerosis with the acute arteriolar lesions of malignant hypertension, both gave the characteristics of "vascular fibrinoid" and the only difference detected between them was the absence of acid phosphatase in the hyalin lesion while it was detected in the necrotic ("fibrinoid") lesion. Both revealed similar microspectroscopic features as obtained by ultraviolet absorption.

3. *Thrombotic Thrombocytopenia.*—Since the early description of this entity, a vascular component in its pathogenesis has been appreciated. Whether the vascular component results mainly from thrombosis or embolism or whether it results from a primary intrinsic vascular lesion has been the source of various considerations. Orbison and co-workers[22,23] and Gore[24] championed a primary vascular lesion. Orbison alluded to the fibrinoid change in the blood vessels. Fisher and Creed[25] more specifically designated the vascular changes as of fibrinoid type.

One of the main vascular disturbances in thrombotic thrombocytopenia results from the development of occlusive masses in small arteries and arterioles. These vessels in turn become distended in an aneurysmal fashion. A compromise of the circulation within the viscera transpires and areas of ischemia and infarction develop. Neurologic manifestations become outstanding. At the same time a hemolytic anemia is present and a fluctuating thrombocytopenia of varying proportions occurs.

Orbison described a similarity between the appearance of the occlusive masses and certain changes within the arteriolar wall.[22,23] Orbison also recognized similarities between the lesions of thrombotic thrombocytopenia and certain vascular lesions of the nephrectomized dog,[19] especially following the explosive vascular disruption attendant on renal ablation plus a high sodium intake.

We have confirmed Orbison's findings in demonstrating lesions in the nephrectomized dog which closely resemble those of thrombotic thrombocytopenia.[18] Moreover, in the latter lesions both the occlusive masses and the arteriolar wall describe the characteristics of "vascular fibrinoid." These two components (luminal and mural) are frequently continuous with each other. Based on other experiences with the nephrectomized preparation alluded to above, we have interpreted these lesions in the dog as derived mainly from vascular smooth muscle.

It is of interest that the occlusive masses as well as the vascular wall in a case of thrombotic thrombocytopenia studied in our laboratory yielded the characteristics of vascular fibrinoid. This suggests that the material in the wall of the arteries and arterioles which appears to give rise to the primary base of the occlusive masses has a derivation similar to that following renal ablation of the dog and in malignant hypertension of man.

4. *Bilateral (symmetrical) Renal Cortical Necrosis.*—This remarkable entity occurs mostly (60 per cent of cases) in pregnancy.[26] Of prominence is ischemic necrosis of the renal cortex which, in turn, is considered to result from a vascular disturbance. The lesion is reproduced experimentally in the Shwartzman phenomenon or by the injection of bacterial toxin directly into the renal artery without a preparatory dose.[27] The details of the pathogenesis remain obscure. Intense congestion of the glomerular and peritubular capillaries occurs.[28] In addition, the fibrinoid change involves the wall of interlobular arteries and occlusive masses of fibrinoid may be seen in the lumen of these vessels and in the glomeruli.[27,29] The fibrinoid in this entity has the characteristics of "vascular fibrinoid."

5. *The Kidney in the Shwartzman Reaction.*[30]—The renal cortical necrosis due to the Shwartzman reaction is associated with the deposition of fibrinoid within the interlobular arteries, arterioles, glomeruli, tubules and peritublar areas of the kidney. In all of these locations the fibrinoid displays the characteristics of vascular fibrinoid. Transitional changes between the intact vascular smooth muscle and the fibrinoid in the media may be observed just as in the experiments on bilateral nephrectomy of the dog. Fibrinoid within the area of the media of interlobular arteries may be limited by a thin membrane on the luminal and in the adventitial sides. On the luminal side this appears to be the basement membrane of the endothelium. The presence of this membranous remnant may be taken to favor a medial origin as opposed to an infiltrative origin. The fibrinoid may replace precisely the area of the media within twelve hours or less following the second dose of bacterial toxin. Continuity between the fibrinoid in the media and polypoid masses in the lumen of the vessel may be observed. The latter are similar to those seen following renal ablation of the dog, in thrombotic thrombocytopenia and in renal cortical necrosis.

The similarity between the vascular fibrinoid of the Shwartzman reaction to that following renal ablation, the seeming transitional changes between normal vascular smooth muscle and this fibrinoid and the confinement to the media in some aretries led us to suggest a major contribution from disrupted vascular smooth muscle to the make-up of this substance.

6. *The Kidney from Diabetic Subjects.*[31]—The lesions of diabetic glomerulosclerosis frequently include two major components: a nodule with peripheral cells and a core having the staining characteristics of collagen, and globular, elongated or crescentic eosinophilic and acellular masses which have the characteristics of vascular fibrinoid. The cellular nodule may be surrounded by dilated capillaries or by crescented masses of fibrinoid. Fibrinoid with the same characteristics as that within the glomeruli is also observed in the wall and lumen of the afferent arterioles, within the capsular space and within the tubules. The impression has been gained that the glomerular, capsular and tubular fibrinoid are derived from the arteriolar fibrinoid by embolization and escape into the tubules.

Fibrinoid Derived from Autolyzed Normal Smooth Muscle[32]

In the above treatment, "vascular fibrinoid" has been described in a group of disorders which not only seem to be initiated by different etiologies but which also express themselves differently from a clinical standpoint. In each entity, the walls of small arteries and arterioles, the lumina of these vessels and that of nearby capillaries may contain the substance designated as "fibrinoid." The concept that this "vascular fibrinoid" is derived largely from vascular smooth muscle has been proposed mainly as a consequence of a series of inferential considerations. The findings upon which this view is based include the location of the fibrinoid material precisely within the media, the disappearance of normal smooth muscle as the material appears, the rapid appearance of the material (a few hours) in some states, the direct continuity of the material with normal appearing media, transitional changes between the normal muscle and the material and certain similarities in the staining, histochemical and microspectroscopic characteristics of the material and normal vascular smooth muscle. In view of the possible relationship between vascular smooth muscle and fibrinoid it was decided to determine whether normal smooth muscle subjected to autolysis and disruption in the test tube develops characteristics of vascular fibrinoid.

For this purpose smooth muscle from the stomach and colon of the dog was obtained under sterile conditions. After autolysis, it was mascerated in a Waring blender and gross particles were removed. The fine particles were centrifuged into a button. Grossly in the test tube, and after smearing on a slide, this material had the appearance, staining and histochemical characteristics of vascular fibrinoid.

By passing the autolyzed smooth muscle through meshes of decreasing porosity it was possible to develop a suspension which passed readily through needles of 23 to 25 gauge. The material was injected into the renal artery of the dog and the kidneys were observed after from three to twenty-four hours (acute phase) and after one to eight weeks (subacute phase).

Acute experiments.[32]—The particles of autolyzed smooth muscle lodged within interlobular arteries, arterioles and glomerular capillaries. Moreover, the material was observed free in the capsular spaces, as casts in the tubules and in the peritubular areas apparently within peritubular capillaries. In all sites the injected material displayed the characteristics of vascular fibrinoid. In most sites of deposition the material failed to evoke an inflammatory response. Occasionally an occluded interlobular artery developed necrosis of its wall superimposed by scattered inflammatory cells (arteritis). Focal or total necrosis (infarction) of the glomerulus also occurred.

Where the occlusive masses were associated with injury of the arterial wall, the media of the injured vessel assumed the characteristics of vascular fibrinoid. Here there were two sources of fibrinoid, that derived from the injected autolyzed smooth muscle and that derived *in situ* from the smooth muscle of the media of the artery.

These acute experiments demonstrated the derivation of "fibrinoid" from smooth muscle and the ability of the material to flow and lodge within the vascular and tubular conduits of the kidney. Indeed, in an occasional experiment the "fibrinoid" could be injected into one renal artery and later observed within the vasculature of the opposite kidney.

Subacute experiments.[33]—Study of the kidneys injected with autolyzed smooth muscle one to eight weeks after the injection added to an understanding of the behavior of this material *in vivo*. In these experiments, one kidney was removed and this kidney acted as the control for future comparison. The remaining kidney was injected and replaced in its bed for one to eight weeks. At various times the kidney was obtained and studied microscopically.

The fibrinoid material had the same appearance after one to eight weeks as it did when observed shortly after the injection. The material was present in small arteries and within glomerular capillaries and in each site it had the characteristics of vascular fibrinoid. Because the characteristics were preserved, we believe the vascular material seen at one to eight weeks was the same as that injected.

Within the small arteries the fibrinoid formed polypoid masses which protruded into the lumen. These were covered by endothelium and usually evoked no inflammation or secondary thrombosis. Within the glomeruli, the fibrinoid had the same appearance but here there was usually cellular proliferation about it.

Remarkable features in the subacute experiments consisted of the development of glomerular lesions. These lesions resembled in some respects those observed in the glomerulus in diseases of man associated with vascular fibrinoid. The major morphologic alterations were:

(a) *Membranous thickening of the glomerular capillary wall resembling "diffuse intercapillary sclerosis" and the "wire loop" lesion.*—The capillary wall or the "intercapillary area" became thickened by the accumulation of a hyaline material. The capillary was patent and retained its endothelial lining but it frequently was narrowed by the thickened wall. On cross-section the capillary displayed the so-called "wire loop" configuration. An occasional lobule appeared to have been destroyed by this lesion.

(b) *Increased cellularity.*—Increased glomerular cellularity assumed several expressions. Small elongated cells accumulated concentrically about the remaining "fibrinoid" globs. Capillaries showing hyaline thickening in their walls at times displayed cells in the wall or what appeared to be increased numbers of endothelial cells. Clumps of cells resembling endothelial cells were seen. In some areas a diffuse cellularity of types seen in certain forms of glomerulitis was evident.

(c) *Nodular lesions.*—Nodular lesions were observed in the center or in the periphery of glomerular lobules. These numbered one to three. The center (or core) appeared to be hyalin and the periphery contained cells. These nodular changes displayed features reminiscent of the lesions seen in diabetic glomerulosclerosis and in certain forms of membranous glomerulitis.

(d) *Thickening of Bowman's capsule.*—The parietal layer of Bowman's capsule displayed hyaline thickening. The epithelium covering of the capsule seemed intact.

The hyaline substance causing thickening of the glomerular capillaries, the hyalin of the nodular lesions, the hyalin which was associated with destruction of the glomerular lobules and the hyalin in the thickened Bowman's capsule failed to reveal the pattern of staining and histochemical reactions characteristic of vascular fibrinoid. This material was eosinophilic and positive (purple) with the PAS procedure. It failed to yield a positive reaction for lipids and for other substances observed in vascular fibrinoid. With Mallory's aniline blue stain this hyaline material was blue, with Masson's trichrome stain it was green and with Mallory's phosphotungstic acid hematoxylin it was orange-brown. From these observations it appeared that the injected autolyzed smooth muscle initiated a reaction within the glomerulus characterized by the deposition of a hyalin different from the usual arteriolar hyalin. There is the possibility that this seemingly secondary hyalin was deposited under the influence of cells within the glomerulus, either the normally present endothelial and epithelial cells or the cells which undergo proliferation.

General Comment

This consideration of the substance designated "fibrinoid," as it is encountered in a group of vascular disorders, has served to emphasize one view as available in published works. In so presenting the subject, it is realized that the matter in its present state is a controversial one. It is encouraging, however, to be aware of different approaches which attempt to develop a better understanding of the origin and significance of fibrinoid as observed in blood vessels.

The ultimate cause and source of the fibrinoid of blood vessels is of more than passing morphologic interest. An understanding of this material may well be necessary for the final understanding of major vascular disorders which remain obscure. In the process of searching for means of developing this understanding it seems important whether the material is an infiltrate of hematogenous origin or a product of autochthonous development.

Should vascular fibrinoid have its main origin from vascular smooth muscle, then emphasis should be toward a better understanding of the fundamental causes of smooth muscle injury.

Assuming that vascular fibrinoid is derived mainly from vascular smooth muscle then it would seem that different etiologies of vascular disease may affect the same morphologic end product. That different basic etiologies pertain in the varied vascular disorders herein considered appears quite likely. Certain of these states are associated with systemic hypertension, others are not; some are localized (renal cortical necrosis) while others are generalized (thrombotic thrombocytopenia); inflammation of the arterial and peri-arterial zones may be prominent in some (the arteritides) while minor or absent in others (malignant hypertension); extravascular disturbances may be outstanding in some (hemolytic anemia and thrombocytopenia in thrombotic thrombocytopenia) but not so prominent or absent in others (Shwartzman reaction). These variations are reflected by varied clinical expressions.

The disorders which emphasize fibrinoid in the arterial wall are also associated with the presence of fibrinoid in an extramural location. Major sites of deposition are: the arterial lumen adjacent to the mural involvement; the lumen of capillaries near sites of arterial involvement; the glomerulus; the capsular space of the glomerulus, and renal tubules. Within renal tubules the material appears to become incorporated in casts. This suggests its ultimate presence in the urine.

In order to explain the presence of fibrinoid in sites away from the arterial wall, the concept of an autochthonous arterial origin of this material must invoke migration of the fibrinoid away from its site of origin. The material appears to possess the property of swelling and spreading. Thus continuity between the material in the arterial wall and that in the lumen, on the one hand, and that in the adventitia, on the other, is often encountered. Migration into the lumen is conceivably aided by muscle fibers remaining near the adventitia as well as by the external elastic lamina, when present. A material prepared artificially from normal smooth muscle flows in the blood stream and lodges at different sites. Of particular interest is the demonstration of rupture through the glomerular capillaries and subsequent lodging in renal tubules.

Certain disorders which emphasize vascular fibrinoid are associated with serious glomerular disease. The glomerular disturbances reflect themselves structurally by capillary or intercapillary thickening (membranous glomerulitis, wire loop lesion), increased cellularity and, in special circumstances, by production of nodular lesions. Focal necrosis of the glomerulus also is encountered. These changes move toward ultimate destruction (sclerosis) of the glomerulus and bring secondary tubular changes. The conditions which are attended by these glomerular alterations may also display the deposition of fibrinoid in the glomerulus. It is therefore of interest that an artificial product of normal smooth muscle which resembles vascular fibrinoid is capable of evoking glomerular alterations which, in turn, resemble alterations encountered in the vascular fibrinoid diseases.

Summary

1. Fibrinoid formation is a structural change which occurs within connective tissues and blood vessels in a variety of disorders. Vascular fibrinoid may be distinguished from connective tissue fibrinoid according to its tinctorial properties.

2. Vascular fibrinoid occurs in a variety of conditions including malignant or accelerated hypertension, eclampsia, diabetes mellitus, the collagen diseases, thrombotic thrombocytopenia and renal cortical necrosis. Vascular fibrinoid emphasizes the presence of fats and lipids, free aldehyde groups of polysaccharide origin, acid esters of polysaccharide origin, free carbonyl and protein-bound (SH) groups and free potassium.

3. Reasons for considering the main contribution to formation of vascular fibrinoid by the disintegration of vascular smooth muscle have been presented.

4. Autolyzed normal smooth muscle displays the characteristics of vascular fibrnoid both in the test tube and in the body.

5. Autolyzed smooth muscle, when injected into the kidneys, appears capable of initiating certain glomerular lesions which include increased cellularity, membranous and nodular changes.

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Purpuras and Associated Thrombotic Conditions

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THE BASIC MECHANISMS concerned with hemostasis are four: vascular integrity, the platelets, plasma coagulation factors, and finally, the anticoagulant and fibrinolytic systems.

In most situations involving refractory bleeding or purpura, more than one of these mechanisms are concerned. Thus, platelet deficiencies and impaired vascular integrity commonly go together, and sometimes coagulation and vascular defects are associated.

In this discussion, attention will be directed to vascular abnormalities and platelet deficiencies with only passing references to disorders involving plasma coagulant and anticoagulant or fibrinolytic factors.

The integrity of small arterioles and capillaries may be altered, in a descriptive sense, by increased fragility or by enhanced permeability. Structurally, the capillary consists of a fibrillary membrane, an endothelial cell layer, and cement substance, which binds the cells together. Functionally, the capillary (besides serving as a partially permeable channel) has retractile, contractile, and adhesive properties which, for the most part, are not well understood.

It is known that the cement substance contains hyaluronic acid, and that ascorbic acid, calcium, and probably vitamin D play roles in its formation.

It has been observed that immediately after injury the normal capillary will retract and constrict. This effect lasts only fifteen to thirty seconds and more prolonged constriction is brought about by a substance released from lysed platelets. This material was formerly thought to be serotonin and serotonin undoubtedly does play a role in vascular behavior. But the platelet factor responsible for capillary vasoconstriction is probably not serotonin, and for the present is best termed simply "platelet vasoconstrictor factor."

Furthermore, capillary vessels respond to the plasma content of histamine, which promotes dilatation and increased permeability, and to the concentration of hyaluronidase at the layer of cement substance, which has a dissolving effect.

It seems to be well demonstrated that the adrenal cortex is concerned with capillary permeability possibly through the mediation of a specific steroid, thrombocytosin, released in response to ACTH stimulation.

Usually, where capillary permeability is affected (as in the purpuras) fragility is also involved. Examples are scurvy, drug sensitization and idiopathic thrombocytopenic purpura. But the converse is not necessarily true. Thus, in hereditary hemorrhagic telangiectasia the vessels may be exceedingly fragile but they do not exhibit greater permeability than is the case with normal endarterioles and capillaries.

It has seemed fitting to approach the subject of purpura from the standpoint of a patient with a bleeding disorder. The points deserving particular emphasis in taking the history and performing the physical examination in a patient suspected of having a bleeding disorder are listed on Tables I and II.

The number and complexity of laboratory examinations required for comprehensive analysis of some hemostatic defects are fortunately not required in the great majority of bleeding disorders. Moreover, relatively simple procedures serve to disclose the general nature of the abnormality and, where applicable, indicate the use of more elaborate and definitive tests. It is, nevertheless, of the greatest importance that exact techniques be carried out with care in the performance of the simplest examinations, since they at best are only roughly qualitative and if improperly done may be grossly misleading. On Table III are shown the most important initial or screening tests as well as some of the more specialized examinations and laboratory approaches useful in particular circumstances.

Proceeding from the general examination of a patient we may consider etiologic classification

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From the Thomas Henry Simpson Memorial Institute for Medical Research, Ann Arbor, Michigan.

TABLE I. HISTORY

Characteristics of Bleeding

How?	Overt; occult; bruisability; other
Where?	Site or source; local; generalized
How Long?	Definite; indefinite; lifetime
Onset?	Abrupt; insidious; unknown
When?	Persistent; recurrent; cyclic
Course?	Mild; progressive; variable; fulminant

Circumstances Associated with Bleeding

Spontaneous:	May be any hemorrhagic manifestation
Traumatic:	Often hematoma, hematocele, or refractory bleeding as from dental extraction or minor injury
Menstrual:	Hypermenorrhea; premenstrual epistaxis or purpura
Orthostatic and Mechanical:	Localized purpura
Factitious and Psychogenic:	Epistaxis; cutaneous, gastrointestinal and genitourinary bleeding

Etiologic Considerations with Respect to Bleeding

Congenital or familial incidence
Drug intoxication or idiosyncracy
Food or other sensitization
Occupational or other contacts
Dietary habits with reference to deficiencies
Symptoms of associated illnesses—almost any systemic disease may have purpuric manifestations

TABLE II. PHYSICAL EXAMINATION

Special Features with Reference to Bleeding Disorders

Kind and site or source of bleeding
Appearance of acute or chronic illness
Developmental abnormalities
Nutritional status
Emotional status
Lymph node enlargement
Splenomegaly—hepatomegaly
Bone (sternal) tenderness

with reference to vascular and platelet abnormalities.

In the matter of thrombosis, as it is related to generalized bleeding disorders, we are concerned with three situations:

1. A chronic, periodic thrombosing tendency associated with excessive numbers of platelets which may be qualitatively deficient, as in the myeloproliferative disorders—chronic granulocytic leukemia, polycythemia vera, essential thrombocythemia and agnogenic myeloid metaplasia.

2. Acute thrombohemolytic thrombocytopenic purpura, a syndrome characterized by acute onset of bleeding, associated with thrombocytopenia, hemolytic anemia, and neurologic manifestations which tend to be migratory and transient, suggesting focal cortical involvement. Hyaline thrombi are present in small arteries of the brain and throughout the body.

TABLE III. LABORATORY PROCEDURES

Screening Examinations

Platelet enumeration or estimation
Capillary resistance test—Rumpel-Leede
Bleeding time—Ivy, Duke
Clotting time—Lee and White
Clot retraction
Lysis of clot
"Prothrombin" time (one stage)

Definitive Examinations

Prothrombin utilization
Thromboplastin generation
Plasma fibrinogen
Ascorbic acid of serum or buffy coat
Special platelet studies
Anticoagulant studies
Special globulin studies
Correction of defect *in vitro*

TABLE IV. VASCULAR ABNORMALITIES

Hereditary and Familial Defects

Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber syndrome)
Vascular Pseudohemophilia (von Willebrand's purpura)
Pulmonary hemosiderosis (idiopathic hemoptysis)
Angiokeratoma corporis diffusum universale (Fabry's syndrome)
Ehlers-Danlos syndrome (mesenchymal dysplasia with cutis laxa, et cetera)

Acquired Defects

Nutritional deficiencies (scurvy, megaloblastic anemias, protein malnutrition)
Allergic sensitization (Henoch-Schönlein purpura)
Drug sensitization
Vasculitis (lupus erythematosus disseminata, polyarteritis nodosa, angio-dermatomyositis)
Infections
Metabolic disorders (renal or hepatic insufficiency)
Tissue hypoxia
Mechanical causes
"Purpura simplex"
Purpura fulminans

TABLE V. PLATELET ABNORMALITIES

Thrombocytopenia—Megakaryocytic

Primary
Idiopathic thrombocytopenic purpura (Werlhof)
Congenital and neonatal thrombocytopenia
Thrombocytopenia with hemolytic anemia (Evans)
Thrombohemolytic thrombocytopenia (Moschcowitz)
Secondary
Acute infections, usually viral
Chronic granulomatous infections
"Collagen" disorders
Congestive and other splenomegalies
Neoplasms, especially leukemias and lymphomas
Drug sensitization reactions (quinidine most important)
Associated with hemangioma in infancy
After excessive bleeding with replacement by multiple transfusions

Thrombocytopenia—Amegakaryocytic

Selective megakaryocytic dysplasia (usually due to drugs, as gold, sulfonamides, et cetera)
Aplastic and hypoplastic anemias
Metastatic and leukemic marrow involvement

Thrombocytopathia

Thrombocytasthenic purpura (Glanzmann's purpura)
Platelet thromboplastin profactor deficiency
Myeloproliferative disorders (granulocytic leukemia, polycythemia vera, agnogenic myeloid metaplasia, essential thrombocythemia)

3. Amniotic infusion with generalized intravascular clotting and resulting defibrinogenation.

The therapy of vascular and thrombocytopenic purpuras may be considered under the following general objectives:

1. Removal of cause when possible, for example, detection and elimination of drug or other allergen, treatment of infection, correction of vitamin deficiency, or eradication of hemangioma by irradiation.
2. Treatment of associated primary disease when not curable, as in case of leukemia, lymphoma, collagen disorders, hepatic or renal disease.
3. Replacement of missing factors supplied by platelets, as by means of fresh whole blood transfusions using siliconized containers, needles and tubing, or by suspensions of separated intact platelets. It is to be expected that preserved platelet fractions or lipids extracted from brain tissue may soon become available for the temporary correction of hemorrhagic disorders caused by platelet deficiencies.
4. Attempts to improve capillary permeability and fragility. This may be done by the administration of vitamin C, and of the bioflavonoids such as rutin, quercetin, hesperidin and esculin; or hormone therapy may be employed, including the use of ACTH, the corticosteroids and the estrogens, such as ethinylestradiol.
5. Attempts to block or diminish the intensity of auto-immune reactions as by the administration of ACTH or corticosteroids, or by splenectomy.

Conclusions

As clinical and laboratory observations on the purpuras have been extended, the complexity of the problem has become more apparent.

With further clarification of the mechanisms of hemostasis which we may expect from the disciplines of biochemistry, physiology, and immunology and from such techniques as protein separation and electron microscopy, it is to be expected that the problems of abnormal bleeding will be simplified and available therapeutic modalities will become more rational and more efficacious.

DISEASES OF SMALL ARTERIES AND ARTERIOLES

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Studies on Plasmin: Factors in Lysis of Thrombi

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THROMBOSIS and the lysis of thrombi are of fundamental importance in physiology and pathology as is so well indicated by this conference. Unfortunately, these reactions are complicated, both in the test tube and even more so *in vivo,* and have been made more difficult to understand by the complicated and reduplicative terminology used. In this discussion, we are particularly interested in the last stage of coagulation, the formation of fibrin by the action of thrombin on fibrinogen, and the lysis of this fibrin gel. *In vitro* fibrin can be lysed by many proteolytic enzymes, but *in vivo* the enzyme primarily involved is plasmin or fibrinolysin. But, what is plasmin?

Plasmin is found as an active enzyme in the blood under certain circumstances: with severe trauma, electro-shock, anxiety, after injection of adrenalin, with cyanosis, and in many other conditions. Under usual conditions, however, it is present as an inactive precursor, plasminogen. This precursor has been shown to be a complex of two substances, a proactivator and a proenzyme.

Using streptokinase, the best activator of human plasminogen available to us, the activation pattern is as follows: proactivator is activated by streptokinase and, *in vivo,* to produce activator, which then acts on the proenzyme (plasminogen) to produce enzyme.[1,2] Unfortunately, the kinetics are not sufficiently clear to establish that streptokinase does not act directly on plasminogen to produce plasmin.

Human blood activator will activate not only human, but also, bovine plasminogen and to a lesser extent, the plasminogen of other species.[3] This is the reason for many of our difficulties in studying fibrinolysis. Although plasmin is proteolytic and will digest casein, fibrinogen, and certain other proteins,[4] we are particularly interested in its specific effect on fibrin. It is practically impossible to obtain fibrinogen and thrombin of bovine or human origin, free of plasminogen.[5] Our tests are, therefore, made on artificial proteins, on contaminated substrates or on complicated substrates such as the patient's own plasma or blood (whole clot lysis). All of these give different results, so our evaluations are qualitative.

Present also in the blood are inhibitors for proteolytic enzymes such as trypsin and chymotrypsin, and for plasmin itself.[6] There are at least two antitrypsins and two antiplasmins.[7] These inhibitors combine with active enzyme in a ratio of 1:1 to produce a fairly stable complex. The supposed activation of plasmin by chloroform and other organic solvents is probably on the basis of destruction of this inhibitor, thus permitting spontaneous activation of the enzyme.[8] Significant increase of the inhibitor occurs after major trauma, operations, in some chronic disease,[9] and the infusion of plasmin[10] and may be of importance in the thrombotic syndrome present after surgery and with some chronic disease.

In the discussion of *in vitro* tests and fundamental concepts, it is also important to mention the significance of fibrinogen concentration on fibrinolysis. Dr. Freiman, in our own laboratory, has recently shown that there is a direct relationship between fibrinogen content of a clot and its rate or ease of lysis.[11] This is of great significance in the dilution techniques of determining fibrinolysis, in the whole clot lysis techniques, and probably in the patient.

I should like to propose at this time the following concept (not entirely original) of hemostasis related to the coagulation of blood. Constantly occurring are thrombosis and fibrin formation, and coincidental lysis. Under normal circumstances this is in balance. Even under nearly normal circumstances injuries to vessels take place,

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but extravasation of blood is prevented by a small platelet or fibrin plug or by a thin layer of fibrin on the endothelium. Indeed, Copley[12] has suggested and Loos[13] has shown by ultra microscopy that fibrin is present on the endothelium under normal circumstances. If the platelet plugs are inadequate or the clotting mechanism is badly disturbed, oozing does occur.

With severe trauma and multiple injuries to vessels, fibrinolysin or plasmin is activated, and when this is excessive, not only are fibrin clots and platelet plugs lysed, but fibrinogen is destroyed, along with the other clotting factors (Factors V and VII) and prothrombin, to a lesser degree.[14] This permits the excessive oozing seen at times in surgical and certain complicated obstetrical cases.[15] It is necessary to stress that excessive fibrinolysis may be very short-lived, yet produce serious consequences.[16]

On the other hand, we find increases of inhibitor, and increase of fibrinogen after surgery and trauma, and in disease states.[9] This results not in a hypercoagulable state which everyone has been seeking but a hypolytic state. In other words, the clot now formed has a higher fibrinogen content and is more resistant to lysis. At the same time the normal lytic activity is prevented by a high inhibitor level. It is not hard to imagine this persistent clot at a point of vascular injury, obtaining continuous accretions and becoming a propagating thrombus.

I cannot resist the temptation to suggest that this mechanism may even be a factor in atherosclerosis. Several pathologists,[17,18] Duquid and others, have suggested that thrombi are incorporated in the wall as atherosclerotic plaques. If this be so, then chronic distortion of this mechanism in the direction of hard clots or elevation of the inhibitor could result in persistent thrombus or fibrin formation along the intima with ultimate inclusion. Greig[19] has shown a decrease in fibrinolysis with high fat feedings and we have confirmed this observation.

It is undoubtedly in the field of treatment of intravascular thromboses that plasmin has its greatest promise at present. Plasminogen can be produced in quantity and is non-toxic. However, the best activator at present available (streptokinase) has been very difficult to produce in a sufficiently non-toxic form. It seems likely that this problem will be solved in the near future, since relatively non-toxic streptokinase has been produced and, it is expected, can be produced in sufficient quantity for clinical use in the near future. Methods of purification after activation of the streptokinase are also being investigated and we hope will bear fruit.

Using the impure materials available to us, we started working on this problem in 1952. We investigated plasmin for its effects on normal animals and found that we could produce a significant lytic activity in the blood stream of dogs and rabbits, without significant pathological changes.[20] Subsequent physiological studies have shown that with quantities sufficient to lyse thrombi, oozing from open wounds occurred, but this was easily controlled by cessation of the plasmin infusion or by closing the wound.[21] With much larger doses we could destroy the animal's fibrinogen and perhaps the AcG and other clotting factors partially, but these returned to normal rapidly. In an animal with very high antiplasmin levels (pig), we were unable to develop fibrinolytic activity.

Throughout our experiments we have had difficulties in finding a satisfactory method of producing thrombi. Originally, we used thrombin, but found the clots formed were not sufficiently stable, many disappeared spontaneously within twenty-four hours, and we lost many control rabbits from pulmonary emboli due to leakage of the thrombin.[21] Also the thrombin contains bovine plasminogen and so, it is conceivable, would be lysed more rapidly than a normal clot.[22] We then used sodium morrhuate and most of our studies are on these thrombi. In rabbits, dogs and cats we found that venous thrombi could be lysed, with adequate amounts of plasmin in the general circulation, (4 mgm. plasmin./kgm./weight.)[22] With arterial thrombi, more plasmin (6 or more mgm./kgm.)[23] was necessary. The reason for this is probably a mechanical one, since tributaries into the vein are common, permitting an attack at many points, whereas with the artery, diffusion through a column of stagnant blood must occur, necessitating a higher concentration in the general blood stream. Another theoretical factor is the role of the endothelium in lysis.[24]

Direct observations of the thrombus during the infusion of plasmin answer many of the questions that arise in one's mind about the method of lysis. The distended vein in the area of the thrombus, first begins to decrease in size and lose its blue color; then one notices a little oozing from the needle-puncture hole used for the instillation of the coagulant. Following this, flow can be seen into the proximal vein, the tips of the thrombus soften and disappear and then the entire thrombus seems to melt away. The entire time from the beginning of the ooze to complete disappearance, is seldom more than ten minutes.

It seems obvious that small segments of thrombus are freed into the circulation, but these have never been of sufficient size to cause any obvious pulmonary embolization, or even observable thrombus in the pulmonary vessels. It is conceivable that the emboli are dissolved in transit or at the time of lodging by the high concentration of plasmin in the blood. Studies with I 131 labeled fibrinogen show increased concentration in the lungs.

In our early experiments, intravenous thrombin was shown to produce death by pulmonary thrombosis in rabbits.[21] A thromboplastic tumor, the Vx2 tumor of rabbits is also known to produce death by multiple pulmonary thrombosis. These two materials were now used to test the efficacy of plasmin for control of pulmonary thrombosis or embolism.[25] Because of the rapid death of the animals it is necessary to give the plasmin immediately after the thrombin or tumor homogenate. In each instance the thromboses were prevented with adequate amounts of plasmin. It may also be of significance that the number of tumor metastases are decreased in the treated animals.

Having satisfied ourselves that plasmin would lyse thrombi in animals, we began to study the effect of plasmin in patients. Despite the lack of reactions in animals, we were reluctant to proceed directly to systemic use in humans. We, therefore, tried the material for local wounds first in 1952 and had success in the treatment of many serious infected wounds and ulcers.[26] More important, is the fact that in over 200 cases, we have had no significant reactions. These include many fresh wounds.[27]

Our first trial of intravenous material in humans was also in 1952. We used plasminogen prepared in our own laboratories and activated with too great an excess of varidase. This, and the next few patients had advanced carcinoma with metastases. Most had some pyrogenic reaction to the varidase used.

Subsequently, the amount of varidase used was decreased to 1000 units, then to 500, and now to 400 units/mgm. of plasminogen.* With these amounts of varidase, reactions have been much less severe and frequent. With the original activated plasmin prepared commercially, reactions were severe, but more recently material has been made available which has minimal pyrogenic effect.**

Studies of the physiological effect of plasmin in humans were carried out as laboratory personnel were available. These were reported previously.[28] There is a significant, but not alarming, fall in fibrinogen, and no significant change in the other clotting factors such as Factor V, Factor VII and prothrombin, with therapeutic concentrations of plasmin. Even with repeated infusions there is significant fibrinolytic activity, and there are indications that this may even increase with repeated injections without increasing adverse reactions or destruction of fibrinogen and other proteins.

The first treatment of a patient for thrombosis was in October, 1953. She had carcinoma *en cuirasse* of the left breast and had had an attempted transnasal hypophysectomy which had to be abandoned because of severe hemorrhage from the carotid artery. Ten days after this operation, the patient developed all the signs and symptoms of a left subclavian and/or axillary obstruction. Eight hours later plasmin was given with marked temporary improvement, including complete relief of pain, pink color returning to skin, warmth and movement of the hand, but there was questionable pulsation. Subsequently, there was regression and amputation became necessary. At postmortem, one month later no thrombus was found.

The first successful case was a seventy-four-year-old white woman receiving radiation therapy for carcinoma of the pancreas, after an exploratory operation. She developed a rapidly progressive thrombophlebitis with severe increasing pain, edema and rubor. Twenty-four hours after the onset with the signs and symptoms still progressive, the patient was given one course of treatment of 3.2 mgm./kilogram. At the end of the infusion the patient had no pain and nor did the pain recur. Her leg decreased in size, although not to normal, during the next seventy-two hours. She was allowed normal ambulation the next day. She survived ten months without evidence of recurrence.

Since the first case, we have treated forty-six patients: twenty-five with venous thrombosis or thrombophlebitis, five of whom had pulmonary emboli; fifteen with arterial thromboses or em-

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*Furnished by Ortho Pharmaceutical Co. through the courtesy of Dr. H. O. Singer and Dr. R. V. Chappel.

**Furnished by Merck, Sharp and Dohme.

TABLE I. LEG MEASUREMENTS IN INCHES OF PATIENT R. G.

Before and After Treatment with Plasmin for Bilateral, Old (More than Six Weeks) Thrombophlebitis

	11-28-56		12-3-56	
	Left	Right	Left	Right
Foot	9⅜	9⅝	8⅞	8⅞
Ankle	8⅞	9¾	8½	8¾
Calf	12¼	13¾	11⅜	12½
Knee	14¼	13¾	12¾	12⅝
Lower thigh	18	20⅛	16¼	17⅝
Upper thigh	21¾	22¼	20¼	21¼

TABLE II. LEG MEASUREMENTS IN INCHES OF PATIENT S. L.

Before and After Treatment with Plasmin for Unilateral Thrombophlebitis

	Left		Right	
	12-28-56	1-2-57	12-28-56	1-2-57
Foot	9	9¼	10⅛	9¾
Ankle	10½	10	12¼	11¾
Calf	11½	11¾	14⅜	12⅜
Knee	13⅞	13½	15¾	14¼
Lower thigh	18	18½	22	19⅛
Mid thigh	22	23	25¾	23½
Upper thigh	24½	24⅜ (1-1)	29¼	25½ (1-1)

TABLE III. LEG MEASUREMENTS IN INCHES OF PATIENT R. K.

Before and After Treatment with Plasmin for Pulmonary Embolus

	Before		After Treatment with Plasmin					
	9-11-57		9-12-57		9-13-57		9-16-57	
	L.	R.	L.	R.	L.	R.	L.	R.
Foot	9¾	9⅞	8½	8⅝	8¾	8¾	8¾	8¾
Calf (mid)	12½	13⅛	12	12⅞	12	12	12	12
Thigh	19⅛	19⅜	18¾	18	18	18½	18	18½

Note that the decrease in size followed the relief of symptoms. It should also be noted that this patient was up and about after the third day of treatment so his edema should have increased.

bolism; and two, each, of central retinal artery thrombosis, cerebral thrombosis and priapism secondary to thromboses. The two patients with cerebral thromboses were treated reluctantly, inadequately, and unsuccessfully. No coronary occlusions have been treated, as such, because of our fear of the reactions to the drug in these patients. However, one patient with probable coronary occlusion was treated inadvertently.

At the beginning, we sought to treat the patients as the animals were treated, with one infusion of 4-6 mgm./kilogram. This was generally (though not universally) unsuccessful because of inability to complete the infusion owing to reactions and for other less obvious reasons. These include the fact that these patients were all ill, with high fibrinogen levels indicating clots more resistant to lysis and many had high inhibitor levels. We were never able to reach a level sufficiently high to result in lysis of an arterial thrombus although several showed marked improvement. At the present time, with venous thromboses and pulmonary emboli, our procedure is to give several small infusions, usually two the first twenty-four hours, then one in each of two or three subsequent twenty-four hours. The last treatment may be delayed forty-eight hours. With this regime, we have had excellent success.

Of the twenty patients with venous thromboses receiving adequate treatment, seven had excellent results. An excellent result implies subjective and objective return to normal. Twelve have had good results. A good result implies subjective and some objective improvement. In no case have other treatments been given to these patients before the result has been determined.

A discussion of a few cases will illustrate what we mean by excellent results. Mrs. R. G. and Mrs. S. L. have been reported previously.[29] The former had bilateral thrombosis with marked edema of six to eight weeks' duration, following a pelvic exenteration. She was bedridden because of general weakness, the leg edema, and a staphylococcus septicemia. Furthermore, practically all her peripheral vessels were thrombosed so that parenteral fluids and blood transfusions were almost impossible to administer. After the first plasmin treatment her veins opened sufficiently for easy intravenous infusion and after four treatments her legs returned to normal size and she was able to be up and about in a walker (Table I). She was discharged to fly to her home in Florida two weeks after treatment and had no recurrence of the thrombosis at the time of her death from recurrent cancer more than six months later.

Mrs. S. L., a patient with unilateral thrombosis and edema of an even greater degree, cleared completely after four treatments (Table II).

Our most recent patient, Mr. R. K., a thirty-five-year-old white man was admitted to Ewing Hospital on September 6, 1957, with acute leukemia of two weeks' duration. He had a platelet count of 39,000 which subsequently fell to 32,000. On the second hospital day he complained of pain in a cord over the right ankle. Twenty-four hours later, he had marked calf tenderness, and a markedly positive Homan sign. The next day he had pain in the left chest with a friction rub. His physicians did not wish to use anticoagulants or consider ligation. Twenty-four hours later, five days after onset, he was given his first infusion of spontaneously activated plasmin, and in six hours a dose of 1.5 mgm./kgm. of plasminogen activated with 400 units of streptokinase as varidase/milligram. After the first treatment, his calf tenderness decreased and after the second, it was almost completely relieved. He had no more chest pain. The chart indicated the improvement as determined by leg measurements (Table III).

This patient makes the fifth patient with a diagnosis of pulmonary embolus or emboli, treated with plasmin. Four had complete regression with plasmin treatment. A fifth patient having all criteria for multiple pulmonary emboli, died of pneumonia and metastatic carcinoma and at postmortem no emboli were seen.

Obviously with arterial thromboses or emboli, this course is impossible and we have used two different methods here. One is to start a plasmin infusion immediately and then remove the embolus at a suitable time. Even with emboli over sixteen hours old, the circulation improves markedly and in one operative case and one postmortem case, the embolus lay free in the vessel, making the removal extremely simple. The other method has been by direct instillation into the thrombus, at operation. Two cases have been reported previously,[29] and we have subsequently had two successful cases at Bellevue Hospital. In only one case have we had success with general infusion.

Discussion and Summary

Therapeutic enzymology is in its infancy, but holds great promise for the future. Plasmin, or fibrinolysin, appears to be the best hope for rapid treatment of thrombotic diseases.

Preliminary studies have shown that it can be given with safety and without serious effect on the animal organism. Pyrogenic reactions at present a serious problem in the human, are undoubtedly due to impurities in the activator. Methods of preparing activator without this disadvantage—either a purified streptokinase, a urokinase, or other activator—are being pressed. It is conceivable that satisfactory methods for purifying plasmin after activation, may be developed even before a better activator is obtained.

Even with the poor and varied materials available, it has been possible to lyse large thrombi, both venous and arterial, of a serious nature in many individuals. However, coronary thrombosis, the most frequent and serious lesion to be encountered, has not been attacked, clinically. There is no reason to suspect that with a nontoxic, nonpyrogenic material this could not be attacked as well. At present, we are investigating the problem of the production of acute thrombi causing infarction in dogs, and the effect on the infarct of this lysis by plasmin, as a preliminary to clinical trials.

The problem of cerebral thromboses or emboli calls for careful investigation. It is conceivable that if the clot were lysed, the sudden return of normal blood pressure to a necrotic area might result in hemorrhage with results worse than the thrombus. We are hopefully awaiting the results of the thorough studies being carried out by Doctor Whisnant, Doctor Millikan, and their group here at the Mayo Clinic.

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The Minnesota Civil Defense Medical Care Plan

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THE MINNESOTA Civil Defense program follows closely the recently revised plans of the Health Office of the Federal Civil Defense Administration. The vulnerability of our target cities to attack with thermonuclear weapons of high potential; the concentrations of population, medical personnel, supplies and equipment, hospitals and industries brings us face to face with the problem of the survival of our nation. For example, an accurately placed 20 megaton bomb, ground burst, without warning, or in the forseeable future—an intercontinental ballistic missile (ICBM)—could destroy both St. Paul and Minneapolis. Approximately one half of Minnesota's physicians, nurses and other health personnel would be eliminated and probably 60 per cent or more of the State's medical supplies, equipment and industrial capacity would be destroyed.

Across the nation similar destruction could occur with the loss of several hundreds of thousands of hospital beds. The sobering realization of the gravity of the national situation made necessary a re-evaluation of the Civil Defense Medical Program. Re-evaluation was necessary to assure the best possible care of non-casualties in order to reorganize industrial production to fight back, in addition to the care of casualties for which most previous planning was directed. The appalling statistics of the National Exercise Alert of July, 1957 (eighteen million dead on the sixteenth day after the attack, thirty-seven million surviving casualties and ninety-five million surviving non-casualties) present a formidable challenge to all health services of the nation. These calculations took into consideration a warning time of two hours, some degree of successful evacuation and/or protection in shelters. The need for intensive medical planning is obvious.

It is becoming increasingly apparent that the care of mass casualties and that of the non-casualty surviving population must primarily be assumed by medical resources in the areas surrounding target cities and that concentration of planning efforts must be undertaken in outlying communities.

The American Medical Association has entered into a contract with the Federal Civil Defense Administration (FCDA) to conduct a study of the medical aspects of Civil Defense. It is to be completed in sixteen months at a cost of $150,000, the funds being provided by FCDA. The study project has been started in the Minneapolis-St. Paul area, and its objectives are:

1. To develop and recommend the planning, training, and operational organization needed as a basis for a National Emergency Medical Care Plan for treatment and care of casualties and non-casualties prior to, during, and after a hypothetical 20 megaton ground burst thermonuclear attack on the United States.
2. To outline the basic role and emergency medical responsibilities of the medical profession in the immediate pre-attack and post-attack period.
3. To outline those functions and responsibilities of the medical profession that may properly be delegated and performed by paramedical personnel under the general direction of the medical profession.
4. To furnish advice and recommendations as to training and education that is needed by all health personnel (professional and non-professional) so that they may be prepared for operational capability in the event of enemy attack.

The AMA Commission consists of six members, two from the Council on National Defense, two from the Council on Medical Services, two appointed by the Minnesota State Medical Association (one representing the Hennepin County Medical Society and one representing the Ramsey County Medical Society) under a staff director, Dr. Earle Standlee. The Commission will seek the advice of the specialty groups of medicine, the allied health professions and other groups that could assist in the conduction of the study project.

Dr. Waldron is also a Doctor of Dental Surgery and a Fellow of the American College of Surgeons. He serves as Deputy Director for Medical Services, Department of Civil Defense, State of Minnesota.

The basic operational facility is the 200-cot (or litter) FCDA emergency hospital (CDEH), or its equivalent in single or multiple units. It is a completely equipped definitive hospital based on a long-range appraisal of post-attack requirements for both casualty and non-casualty medical care. Such facilities would be located 50 miles or more from an anticipated aiming point in the critical target areas of the Twin Cities and Duluth. One of these assembled packaged hospitals is located at Glen Lake Sanatorium, set up as a streamlined display for demonstration and training. A second is located at Moose Lake for training and twelve more are stored in Minnesota institutions. We hope to receive many more; twenty are now on order. It is obvious that FCDA can supply but a relatively small number of the total number of 350 improvised hospitals (70,000 beds) indicated for Minnesota. The staffing pattern for a CDEH includes eleven physicians, six dentists or veterinarians, thirty-three registered nurses and five anesthetists. Trained aides including 100 nurses aides, clerks, messengers, administrative and maintenance staff and helpers increase the personnel to a total of 300 for each CDEH. This staffing pattern is an organization guide for the expansion of Twin City and Duluth hospitals after their site-to-site relocation to cities designated by the Minnesota-Wisconsin FCDA Survival Plan Project. This planning provides for the operation of 35,000 beds and in addition provides a reserve pool of several hundred physicians and dentists for assignment in communities with hospital expansion plans throughout Minnesota. It is obvious that an attack without warning or lack of sufficient time for evacuation would largely cancel out this large potential medical care resource for Civil Defense.

All Minnesota communities with hospitals have made plans to increase the bed capacity of the local hospital as much as possible, and in addition to provide building facilities to make possible a total expansion up to 10 times or more the existing number of hospital beds. For example: City X, population 2,500, a forty-bed hospital and five physicians, is expected to increase its hospital capacity to eighty or more beds and select schools or other suitable buildings for 300 or more additional cots or beds. The staffing pattern for professional workers of the 200-cot CDEH used as a planning guide, calls for eleven physicians, six dentists, two pharmacists, five anesthetists and thirty-three registered nurses. The remaining personnel: 100 auxiliary nurses aides, eighteen trained aides, three first aid technicians, thirty-six clerks, fourteen messengers and sixty-four helpers, make a total of approximately 250 persons. Twice this number would be indicated for City X. The State Office of Civil Defense would endeavor to assign seventeen physicians to assist the five physicians of City X and other professional deficiencies not found by local and county surveys. The Civil Defense director of City X, the hospital administrator and staff, nurses and other groups are responsible for recruiting, training and organizing most of the personnel. By so doing, the Civil Defense Welfare Service of City X would be relieved of the necessity of providing housing and food for up to 500 persons needed for the operation of the 400-bed hospital facilities if brought in from other communities. The State Department of Civil Defense will endeavor to assist all hospital communities in their organization plans. Specific information is being sent to all hospital cities.

The Department of Civil Defense has divided the State into five Mobile Support Areas (MSA), each under an area commander to co-ordinate all CD activities, as follows:

MSA I.—South of the Twin Cities, to support St. Paul.

MSA II.—The northeastern section of Minnesota, to support the Duluth Target area.

MSA III.—The northwestern section of Minnesota, to support the secondary target areas of Fargo-Moorhead, Grand Forks-East Grand Forks, and Manitoba, Winnipeg.

MSA IV and V.—Southwest, west, northwest and north of the Twin Cities, to support Minneapolis.

In these areas, communities with hospitals further distant than the cities selected for site-to-site relocation of target city hospitals are to plan for the expansion of hospital facilities. In MSA IV and MSA V they are expected to plan for 17,200 beds, the staffing pattern for which requires 950 physicians. There is need, also, for about 700 additional physicians to be assigned by Civil Defense to reinforce the supply of local physicians. Similar shortages are estimated for MSA I and MSA II. Such calculations confirm the estimated critical shortage of physicians that would occur after a mass attack on American target cities.

Surveys being made by nurses' organizations of registered nurses (active, part-time and retired) though still incomplete, would seem to show that an adequate number of R.N.'s will be available. Short courses (seven lessons of two hours) on atomic nursing are being given and will be available throughout the state. Eleven thousand five hundred registered nurses are required to staff a 350 CDEH (the equivalent of 70,000 beds).

Auxiliary nurses, students, licensed practical nurses, aides, and Red Cross home nursing trainees present a great problem of large deficiencies in numbers to meet the requirements of the Emergency Treatment Stations (ETS) and emergency hospitals. Every effort must be made to locate persons with auxiliary nursing training. The Red Cross is making every effort to step up the training in home nursing to meet this personnel deficiency. Junior and senior high school girls are a potential source of trainees in home nursing as well as college students in communities with colleges. Thirty five thousand auxiliary nurses are required to staff 350 emergency hospitals or 70,000 beds.

An outstanding development in the over-all Civil Defense organization for the target cities of Minneapolis and St. Paul is the UNICOM Operations Plan authorized by the Governor and Executive Council of Minnesota. It extends the authority and co-ordinating functions of the Civil Defense Director of St. Paul for a distance of 50 miles from an estimated inter-city aiming point over a half circle area (UNICOM DIVISION I), east of U.S. Highway 65. A similar half circle of a 50-mile radius west of U.S. Highway 65 (UNICOM DIVISION II) is under the direction of the Civil Defense Director of Minneapolis. The inner portion of the UNICOM area is the target area with a diameter of fifty miles allowing for a five mile error in the aiming point. The target area consists of four rings: The "A" ring of complete destruction has a radius of 5 to 7 miles; beyond this for 3 to 5 miles, the "B" ring presents such extensive damage that all building must be torn down; the "C" ring extends for an additional 3 to 5 miles in which practically all buildings must be vacated for repairs; in the "D" ring, damage will be of varying degrees but most buildings could be occupied while being repaired.

Extending for 25 miles past the target area is the assembly area of UNICOM—that of Division I extending 25 miles into Wisconsin. Most of the Emergency Treatment Stations (ETS) will be set up in the assembly area.

Under a 20 megaton surface burst attack, blast and thermal destruction may extend 11 or more miles from the point of detonation to doom all unprotected individuals. Casualty survivors may be found between the 11-mile and 17-mile radius —the outer portion of the "C" ring and the "D" ring. Such survivors may be exposed to initial and residual radiation, making it improbable that they will be rescued and receive medical care at the Emergency Treatment Stations within the time that attention could be life-saving. Beyond the area of blast and thermal damage, a megaton burst may produce serious radiation intensities for a few miles up-wind. Every effort should be made to provide radiation reduction protection. An average of nine hours and thirty minutes must elapse before radiation intensities decay to levels safe for continuous medical operations.

The Early Care of Bomb Casualties

Emergency Treatment Stations (ETS) replace the station group, which up until recently has been designated the casualty first aid system. Each ETS would be located on main Civil Defense evacuation highways as close to the damage area as permitted by radiation monitoring calculations. The selected locations are 30 to 50 miles from the zero point, but if indicated might be set up several miles closer in or moved out, if necessary, as it is a mobile facility. The function of the ETS is life saving medical and surgical care. It is a 128-litter hospital with provision for an additional 100 litters for holding. The basic equipment and supplies are the same as previously published (FCDA Technical Manual 11-1) for the casualty first aid services, and duplicates those of the thirty first aid units stored in Minnesota cities. The ETS does not have a clinical laboratory nor x-ray facilities. It is anticipated that an ETS will operate around the clock for at least one week and continue operation for a total of three weeks. Schools or other suitable buildings with 10,000 sq. ft. of floor space will be used. In certain favorable locations (adequate buildings, safety from fallout, et cetera), an ETS may later be converted into a 200-cot (or litter) definitive hospital, with the addition of more equipment. Associated with the ETS will be a first aid, receiving and sorting section, to admit casualties requiring immediate care at the ETS; to sort and to arrange transportation to definitive hospitals of casualties requiring further care. Indicated first aid and other care will be instituted at the ETS.

The ETS staffing pattern includes eight physicians, six dentists and ten registered nurses, and the ETS for the Twin Cities will be located in twenty-three communities in Minnesota and Wisconsin. Most of the auxiliary nurses, other paramedical and maintenance personnel will be recruited, trained, and organized in the selected ETS communities. The medical, dental, and registered nurses personnel deficiencies will largely be supplied from suburbs close to the target areas if available. If not, such personnel will be sent forward from hospital cities 50 or more miles from the area of detonation.

Attached to each ETS are the Forward Aid Area rescue groups consisting of ninety-six litter-bearers (twenty-four teams of four) and twenty ambulance drivers—two to each vehicle—all trained in first aid. In case the selected ETS community does not have sufficient population to organize these groups, other communities on the same evacuation highways will organize the litter-bearer ambulance groups. It should be noted that to conserve professional workers for the staggering needs of mass casualties, no professionals, physicians, dentists, veterinarians or registered nurses will be assigned closer to the damage area than the ETS.

The blood program for Minnesota is making good progress with the plan of storing one year's supply in outlying hospitals to be replaced as used. An intensive training program of technicians is being organized. The estimated requirements for blood is 1500 units per 1000 casualties, during the first three weeks. FCDA, the Red Cross, and other blood banks are combining to produce 300,000 units of serum albumin per year for emergency.

Equipment and Supplies

FCDA has stored in forty warehouses, sufficient equipment and supplies to care for more than 3,500,000 casualties for three weeks and for more than a million non-casualties for a comparable period. Warehouses are located 30 to 100 miles from targets. Replenishment packs are being assembled—such as a three-weeks unit for the CDEH, 1000 casualty packs for the ETS, and 100 casualty packs suitable for civilian disasters. Recent plans are under way to assemble equipment for specialized surgery—dental, gastrointestinal, genito-urinary, cardio-thoracic and eye-ear-nose-throat, to meet the needs of long range care of our citizens. Improvisation must of necessity be of prime importance for Civil Defense Medical Care. Fortunately, drug stores, hardware and general stores, physicians and, where present, the hospital of a community, can provide enough equipment and supplies to treat at least 500 casualties. The concentration of most of our vulnerable medical equipment and supply industry is a matter of deep concern. Unless its dispersal can be accomplished, national stockpiling for the needs of a surviving population for 272 days post-attack is considered necessary by FCDA.

Dr. Max Van Sandt, Director of the Health Office, FCDA, stated at the eighth County Medical Societies Civil Defense Conference in Chicago, November 9-10, 1957:

> "Under thermonuclear attack with ground burst weapons in the megaton range under present capabilities because of production vulnerability, minimum stockpiles and small inventories from production source to the user the recommended priority for medical care is:
>
> 1. Maintenance of health and production capability of the non-casualty survivors.
>
> 2. First aid and medical-surgical attention to the minimal (walking wounded) casualties to return them to productivity as soon as possible.
>
> 3. Medical care to women of child-bearing age and children.
>
> 4. Casualties that reach the medical channels.
>
> "In conclusion, planning in the health field must provide for:
>
> 1. Programs that will reduce casualties and, where possible, reduce loss of existing facilities.
>
> 2. Medical care to casualties and emergency facilities to replace those estimated to be lost.
>
> 3. Medical care to the non-casualty survivors again in emergency facilities.
>
> 4. Public health protection in a sanitary environment that will allow our efforts toward recovery to function with a minimum loss of personnel to sickness and injury during the post-attack period.
>
> 5. Preparation of our citizens psychologically for the potential post-attack conditions which can be unleashed by an enemy capable of delivering such an attack.
>
> Only in this way can we assure a minimum of health services to the survivors that will give our country a fighting chance to recover from the weight of an attack within the capability of our potential enemy."

Epilepsy in Childhood

M. G. PETERMAN, M.D.
Milwaukee, Wisconsin

IN 1925 a definition for idiopathic or essential or cryptogenic or hereditary epilepsy was proposed by me, as follows: Idiopathic epilepsy is a chronic disease of the brain, characterized by periodically recurring convulsions; lapses, or abnormal mental states occurring in an individual with an inherited cerebral defect; but with no demonstrated pathologic lesion.[1]

Except for the amplifications resulting from progress in the study of this disease, the definition remains an all-inclusive description of this neurologic syndrome. The neurologists have challenged the term idiopathic, but whenever they wish to describe the disease, they use this or a similar term in quotation marks. Many investigators have challenged the hereditary origin of the disease. One in particular stated that the disease, epilepsy, is not inherited, only the tendency is. He also stated that epilepsy was not inherited any more than is diabetes. That is my contention. In my experience with this disease in children I have been able to obtain a positive family history in over 50 per cent of the cases. I have also been able to obtain abnormal electroencephalograms in at least 50 per cent of the near relatives of the patients with epilepsy.

The greatest contribution to the study of this disease came from Professor Hans Berger when he introduced the electroencephalograph in 1929.[2] Until that time, the diagnosis of idiopathic epilepsy was made on the history and a description of seizures, alone. There was no objective evidence to confirm the diagnosis. We were obliged to rely on the history as given, a description of the seizures. and the response to treatment. The diagnosis of idiopathic or cryptogenic epilepsy was made when all organic causes of the convulsions could be excluded. The electroencephalograph has made it possible to confirm the diagnosis and to distinguish the various manifestations and types of the disease. This is extremely important because of the modern advances in treatment.

Today, we do not treat the disease *per se*, rather we treat the specific manifestations of the disease and try to control the potential symptoms. However, the physician who is not familiar with the electroencephalograph must be advised of its limitations. It is a laboratory aid and not a diagnostic machine. Like the x-ray and the electrocardiograph, and all other laboratory aids, it may be of inestimable value in confirming a diagnosis with objective evidence; or, if it is negative, it must not be used to exclude a well-founded clinical diagnosis. The result of a test should never be allowed to confuse the issue. Unless the tracing is positive and definite, it should be interpreted without imagination.

Bucy of the Illinois Neurologic Institute warns his colleagues that, "However, the electroencephalograph is not, independently, a trustworthy diagnostic instrument. No one, by this means alone, can make a satisfactory diagnosis of epilepsy, a brain tumor, or a traumatic injury of the brain. The electroencephalograph by itself is also inadequate evidence on which to base treatment or to render a medical legal opinion. It is a matter of common experience that the information gained from electroencephalographic tracings may be grossly misleading and that great care must be exercised in correlating such records with all other clinical and laboratory data and in evaluating the electroencephalograph accordingly."[3] Boshes in a recent article called attention to the ridiculous extreme to which some of the electroencephalographers have gone in the interpretation of results using this experimental apparatus.[4]

In my definition, "convulsions" signifies the well-known and recognized major seizures of epilepsy. About one-half of the children have their onsets between eight and twelve years of age, usually with a major grand mal convulsion. One-half of these have a preliminary aura. These children seldom develop petit mal subsequently. One-half of the children have their onsets with petit mal or minor seizures which do not have an aura or loss of equilibrium. Most of these children have po-

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Read before the Northwestern Pediatric Society, Saint Paul, Minnesota, February 8, 1957.

tential grand mal seizures and about half of them develop major seizures some time later. The "abnormal mental states" described in my definition are now understood to be either psychomotor epilepsy or hypothalamic syndrome epilepsy.[5] These conditions comprise a significant proportion of the behavior disorders in childhood, particularly between the ages of ten and eighteen years. They may or may not be associated with major convulsions. The diagnosis in psychomotor epilepsy may be confirmed when the electrogram shows positive negative spikes, particularly in the temporal lobes. The hypothalamic syndrome epilepsy may be confirmed when the electrogram shows fourteen and/or six-per-second positive spikes in the temporal lobe.[6]

The most effective treatment for epilepsy ever devised has been fasting and purging described by Hippocrates.[7] An attempt to reproduce the chemical effects of fasting resulted in the introduction of the ketogenic or high-fat diet.[8] This diet, like the diabetic diet, requires weighing or measuring of the food intake. It is no more undesirable or objectionable than the diabetic diet. Since it is aimed at the correction of an inherited serious disease which tends to be progressive and eventually produces deterioration of the brain, it is worth a fair trial. The effectiveness of the ketogenic diet can be determined within one week on a fast. If this fasting diet produces ketosis and a tendency to acidosis and does not control or prevent further seizures, the ketogenic diet will not be effective. The ketogenic diet eliminates the abnormalities in the electrogram.[9] It is effective in about 50 per cent of the cases and in all manifestations of the epilepsy syndrome, particularly petit mal and grand mal. If the diet is not practical or feasible, the second choice in treatment is, of course, drugs.

The present status of drug therapy was very well presented by Yahr and Merritt.[10] For the treatment of major convulsions of idiopathic epilepsy or of organic origin the drug of choice is phenobarbital U.S.P. This drug is universally available, relatively inexpensive, and harmless. The drug should be given to anticipate the expected seizures by several hours. The dosage should be increased until the seizures are under control unless undesirable side effects develop. The drug is available in capsules which provide a sustained and continuous release. If phenobarbital in adequate dosage is not effective or if it produces too much sedation or drowsiness, Metharbutal or Gemonil® may be used. This barbiturate is an anticonvulsant with very little sedative effect and no other serious side effects. Phenobarbital is a drug which has diagnostic as well as therapeutic value. If the major convulsions cannot be controlled with an adequate and well-timed dose, the convulsions are probably of organic origin. If the convulsions are increased and misbehavior is aggravated, psychomotor, or hypothalamic syndrome epilepsy should be suspected.

Once the seizures are under control, the drug should be continued in the same dosage for at least one year and until a negative electrogram is obtained. Then the dosage may be tapered off gradually during the following year. The second drug of choice in the treatment of major convulsions is primadone or Mysoline®. This drug is relatively nontoxic and in small and increasing doses may be given to effectiveness usually without undesirable side effects. If neither phenobarbital nor Mysoline® are effective, perhaps a combination of the two will produce the desired results; if not, the next drug to be tried is phenacemide or Phenurone®. This is a toxic drug, but it is given as a calculated hazard in a serious or desperate situation. It is sometimes effective in the control of grand and petit mal seizures. When phenacemide is given, the blood count and the urine must be watched for toxic effects.

Dilantin® has not been a satisfactory drug in children. The therapeutic dose equals the toxic dose and almost always this drug produces disequilibrium and hypertrophy of the gums. A new hydantoin preparation, Peganone®, has now been introduced. I have used this drug for five years and have observed no serious toxic side effects. It is most effective in grand mal.

The diagnosis of petit mal or minor epilepsy should be confirmed if possible because petit mal *variant* seizures are usually of organic origin and do not respond well to the few drugs which are effective in the treatment of petit mal. The two effective drugs for petit mal are Tridione® and Paradione®. Whenever Tridione® or Paradione® are used, phenobarbital should be given concurrently because the diones may precipitate major seizures; also, the blood count should be followed regularly because of possible depression of the bone marrow. The "snow blindness" or visual disturbances which sometimes follow the use of these drugs is not serious and will clear when the drugs are discontinued. If the diones do not control petit mal seizures, phenacemide should be given a trial. With this drug it is not necessary to add phenobarbital since phenacemide is an effective drug in the control of major seizures. Of the various other drugs advocated for the treatment of petit mal I have not found any consistently effective. A recent article reporting effective results with a new compound is open to question. The author admits that he has not effected changes in the electrogram. I have tried all the other drugs in adequate amounts for sufficiently long periods of time to be convinced of their inadequacy.

The treatment of psychomotor seizures and hypothalamic seizures presents a serious problem. Phenobarbital may be given for a diagnostic test. It usually aggravates or precipitates the misbehavior. The first drug which has ever been effective in the control of some of the children with psychomotor epilepsy is phenacemide. This is a toxic drug, but is well worth a trial. In older adolescents and adults it may precipitate a serious psychosis. In some patients it depresses the bone marrow, and in others it may produce liver damage. If the drug is not effective or if signs of toxicity appear, amphetamine (Benzedrine®) may be given a trial. It is a paradoxical fact that Benzedrine® sometimes has a sedative effect in these patients. So far, I have not found any of the antitension drugs of value in the control of behavior in patients with this type of epilepsy.

Surgery on the temporal lobe has not as yet produced any striking results. It is possible that decortication or ablation will help to control the behavior. However, in some reports ablation of the tip of the anterior temporal lobe in which the focus was located with the electroencephalograph has been followed by the appearance of a new focus in the opposite temporal lobe.

Summary

The treatment of epilepsy is a problem for the general practitioner and the pediatrist. The neurologist may be helpful in eliminating organic disease. It will be simpler for all of us if we confine the term "epilepsy" to the idiopathic type in which organic lesions have been excluded. It would be advisable to confine electroencephalography to the larger diagnostic centers, where the latest equipment and the best qualified technicians are available to produce records to be interpreted only by qualified electroencephalographers. There are a limited number of effective drugs available for the treatment of the various types of the epilepsy syndrome. These drugs should be used alone or, if necessary, in combination in an attempt to attain the desired results. The literature is full of reports of spectacular results obtained with new drugs. Many of these drugs have now been discontinued. Therefore, all reports must be carefully evaluated. In many cases the drug treatment resolves itself into a procedure of trial and error.

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HEART DISEASE AND HEART ATTACKS

Heart disease, now one of our greatest health problems, costs the U. S. government over $700 million annually. Loss in working time caused by the disease amounts to approximately $3 billion per year. The total annual outlay of the major sources of research support is currently estimated at $33,672,725.

Though heart attacks are often associated by laymen with over-exertion, they are far more likely to occur during periods of rest. Over half of the victims of coronary heart attacks are stricken while resting or sleeping. Less than 2 per cent are afflicted when engaging in "sports, running, lifting or moving a load."

—*Patterns of Disease*

Clinical-Pathological Conference

Sponsored by
The Minnesota Society
of Clinical Pathologists

CASE PRESENTATION—ST. MARY'S HOSPITAL (DULUTH)

V. A. HARRINGTON, M.D.
Duluth, Minnesota

Clinical History.—This white, single, bricklayer, aged forty-three, was admitted on August 14, 1955, complaining of chest pain. This had begun five days previously; it was in the left chest and was aggravated by coughing and deep breathing. The pain was accompanied by persistent cough productive of a small amount of whitish sputum without blood. Weakness, dyspnea, chills and cold sweats also had been present most of the week. A 25-pound weight loss during the preceding three months had been associated with distinct anorexia.

A review of systems contributed no information other than that he had had three operations on his back five years previously following an injury and culminating in a spinal fusion. He had smoked one package of cigarettes per day for many years; a history of heavy alcoholic intake (at least one pint of whisky per day) was obtained, and alcoholic debauches lasting several days were common several times per month, and most recent immediately antedating the onset of his illness. No family history of tuberculosis could be obtained.

Examination revealed a temperature of 103° F., a pulse rate of 96 beats per minute, a respiratory rate of 24 per minute, and a blood pressure of 110 mm. of mercury systolic and 75 diastolic. The pupils were small, reacting slightly to light. The ocular fundi showed minimal sclerotic changes. The teeth were carious, with gingival infection; evidence of recent dental extractions was noted. Excursions of the left side of the chest were limited, and gross impairment of tactile and vocal fremitus was present over the left lower lung field. Numerous rales were present posteriorly on both sides, and bronchial breathing was noted in the left midlung field. The borders of the heart appeared within normal limits, and the rhythm was regular. The liver was barely palpable beneath the rib margin but was not tender. No other organs or masses were palpable in the abdomen, and tenderness was absent. The remainder of the examination contributed no positive information.

The leukocyte count was 19,350 cells per cubic millimeter (87 per cent neutrophils and 13 per cent lymphocytes), and the value for hemoglobin was 12 gm. per 100 ml. of blood; blood urea nitrogen measured 8 mg. per 100 ml.; the icterus index was 8.7 units. Other values were: serum sodium, 124 m.Eq. per liter; serum potassium, 3.3 m.Eq. per liter; serum albumin 3.3 gm. per 100 ml., and globulin, 2.9 gm.; thymol turbidity, 3.7 units; cephalin flocculation, 2 plus in twenty-four hours and 4 plus in forty-eight hours. A Kline test for syphilis gave negative results. The initial chest roentgenograms showed several areas of increased density in the right lung and a similar density involving the middle and lower portions of the left lung field. The sputum contained Gram-positive cocci in pairs on smears, while the culture showed alpha hemolytic streptococci predominating, with only a few pneumococci. Smears showed no acid-fast bacilli. Mantoux tests (dilutions of 1:10,000 and 1:1000) gave no reactions. An electrocardiogram was not abnormal. The urine had a specific gravity of 1.006, and was negative for albumin, sugar or abnormal microscopic elements.

Treatment with penicillin (600,000 units every six hours) and erythromycin (200 mg. every six-hours) was initiated. During the next twelve days, the patient showed continuous fever, usually between 103° and 105° F. Impairment of cerebration was manifested by recent memory loss, irrational behavior, slurred speech and episodes of frank confusion. The physical and radiologic signs in the lungs persisted and progressed. Blood-tinged sputum appeared. The addition of streptomycin and tetracycline failed to produce any permanent detectable change in the course of his illness, and on August 26, 1955, he was transferred to a tuberculosis sanatorium.

Upon arrival at the Sanatorium, the patient appeared moribund. A sister confirmed the previous history, adding the observation that the patient had had a nonproductive cough for eight or nine months that had become worse suddenly five days before hospitalization. Past employment had included jobs in the merchant marine, steel plants and construction crews. He was born in Minnesota and had left the state only for about three years while in the merchant marine during World War II, which included service in California and the Pacific Ocean.

Physical examination in the sanatorium showed signs of severe dehydration, with fever, toxicity and delirium. Radiologic changes in the chest were similar to those noted previously, although progression of involvement became apparent. The leukocyte count was 20,350, and the hemoglobin measured 11.7 grams. The urine showed a specific gravity of 1.025, 1 plus albumin, no sugar, 10 to 12 leukocytes per high-power microscopic field and occasional erythrocytes. The blood urea nitrogen was 19 mg. per 100 ml., and the serum chlorides were 83 m.Eq. per liter. Because of severe cyanosis, tracheotomy was performed and oxygen administered, with only moderate relief. Numerous smears of sputum showed no acid-fast bacilli, and cultures showed only coagulase-negative staphylococci. Cultures of sputum and tracheal washings for acid-fast bacilli and fungi were not completed before death, but malignant tumor cells were not demonstrable. Treatment with streptomycin, isoniazid and tetracycline, later supplemented with chloramphenicol and sulfadiazine, had no demonstrable effect. Fever continued, delirium passed into coma, cyanosis became constant, and the patient died on September 9, 1955, in an apparent state of respiratory insufficiency approximately one month after the acute onset of his illness.

Discussion

DR V. A. HARRINGTON:* The problem in this case is essentially that of a man with cough, weight loss, rapid progressive pulmonary consolidation, delirium and death. Since the roentgenograms show a con-

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From the files of the Department of Pathology, St. Mary's Hospital, Duluth, Minnesota.

*From the Duluth Clinic.

siderable degree of pathologic changes, they will be presented first.

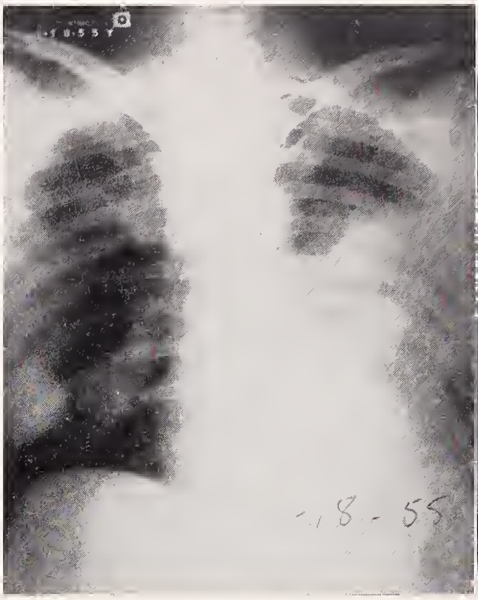

Fig. 1. *(Left)* Roentgenogram four days after admission. Extensive nodular involvement in both lungs is obvious.

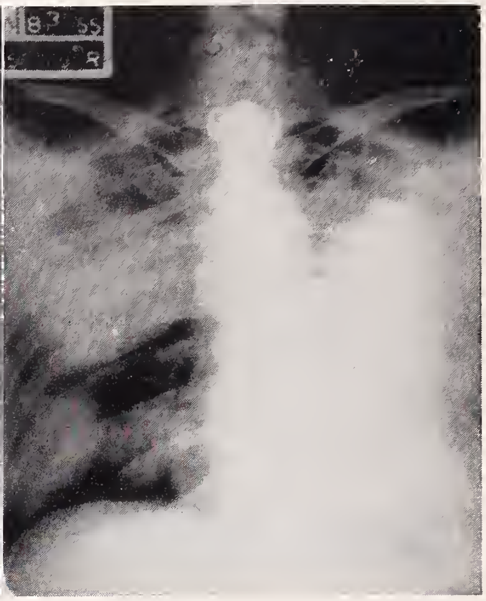

Fig. 2. *(Right)* Roentgenogram seventeen days after admission. Pulmonary involvement is much more extensive.

DR H. G. MOEHRING:* Several larger and smaller densities are obvious in each lung and are not segmentally arranged (Fig. 1). Abnormalities of the thoracic cage are not apparent. The later films show an increase in size of the densities, particularly in the right upper lung fields (Fig. 2). Small zones of decreased density also are noticeable within these densities, suggestive of cavitation, and a fluid level is distinct in some such areas. The last films show little functioning parenchyma. The lack of segmental arrangement of these densities suggests a blood-borne process that could be either inflammatory or neoplastic. Rapid progression makes inflammation more likely and the evidence of cavitation indicates necrosis.

DR. HARRINGTON: Of the malignant tumors, an extremely malignant lymphoblastoma is about the only primary type that would produce such a fulminating picture. The remarkable rapidity of the course, in addition to the fact that the disease remained limited to the chest, makes this diagnosis improbable. A shower of tumor emboli from a hypernephroma coincident with pulmonary infection conceivably could produce this picture, but such an improbable coincidence is unlikely. The clinical course is too rapid to be explained on the basis of metastatic carcinoma, so I consider that malignant tumors of any type can be excluded with reasonable safety.

Multiple pulmonary emboli must be considered but, if these huge densities on the roentgenogram represent infarcts, one would expect that one of the emboli would have caused earlier death. Chronic pulmonary embolization usually results in large numbers of tiny emboli, sufficient to produce pulmonary hypertension and death from failure of the right side of the heart.

Infection appears to be the only logical explanation of the pulmonary pathologic changes. It is inconceivable that this degree of cavitating pulmonary tuberculosis could exist without easily demonstrable acid-fast bacilli in the sputum. The increased leukocyte count, fever and early hypochloremia and hyponatremia all suggest an acute bacterial infection. Among organisms that commonly cause lung abscesses, *Klebsiella pneumonial* (Friedländer's bacillus), staphylococci and streptococci always must be kept in mind. Friedländer's pneumonia, however, usually is distinctly lobar in distribution, the bloody gelatinous sputum is characteristic, and organisms are abundant in the sputum. The acute form of this disease usually kills in less than a week. Streptococcic lung abscesses are usually due to group A, beta hemolytic streptococci, not the alpha hemolytic form isolated from this patient. Pneumococci, which causes 95 per cent of all pneumonias, were scarce in the original sputum smears; in addition, the distribution in the lung is atypical for this organism. Staphylococcal lung infections commonly produce necrosis and abscesses, and are often preceded by dental extractions, as in this patient.

A probable important feature in this patient is chronic alcoholism, for such patients are known to be susceptible to infection and resistant to treatment. In view of the fact that this patient's acute illness was preceded by dental extractions, it is tempting to postulate resulting bacteremia by any of the above-mentioned pyogenic bacteria. This may have been controlled partially by antibiotic therapy, as evidenced by a temporary decline in the temperature curve at that time. The subsequent increase in fever could be explained by secondary invasion by an organism resistant to the antibiotics used. The coagulase-positive strain of staphylococci cultured from the sputum at this time is probably significant, and perhaps the organism was more virulent than usual because of the deteriorated state of the patient resulting from alcoholism. Institution of chloramphenicol therapy (to which the organism had demonstrated sensitivity by *in vitro* tests) was again followed by a drop in the temperature curve, suggesting further that the staphylococci were playing a major role in the inflammatory process at this time and that sterilization of the pulmonary abscesses probably occurred. The final rise in temperature, which persisted to death, probably reflects invasion of a third organism, most likely a fungus.

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*From the Duluth Clinic, Duluth, Minnesota.

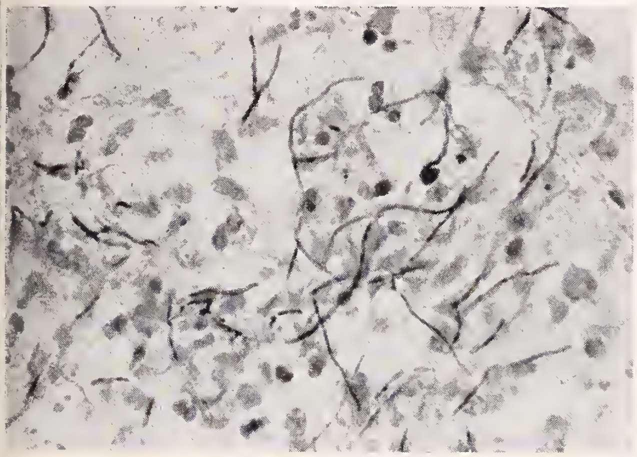

Fig. 3. Section of lung. Large numbers of organisms are apparent (Gram's stain; x900).

A variety of fungi might be expected. *Candida albicans* is the most universally distributed member of the group. To indict it as a cause of pulmonary infection is a difficult clinical problem, one that is hazardous unless the fungus is grown in pure culture, since it is often found incidentally in sputum cultures. In this case, the wide range of antibiotics administered, as well as the fact that no other significant organism was cultured at this time, makes this a likely possibility.

The cerebral changes described can be explained on the basis of toxicity from the overwhelming infection obviously present.

In summary, my reconstruction of the sequence of events would begin with dental extractions, bacteremia, diffuse pyogenic pneumonitis with multiple pulmonary abscesses and cavitation (staphylococcic), antibiotic sterilization and subsequent pulmonary invasion by a fungus (probably *Candida albicans*), progressing to a fatal outcome through pulmonary destruction and respiratory insufficiency.

Dr. Harrington's Diagnosis

Diffuse pulmonary moniliasis following staphylococcic pneumonitis caused by bacteremia induced by dental extractions.

Anatomic Diagnosis

Diffuse pulmonary nocardiosis.

DR. A. C. AUFDERHEIDE: The significant findings at necropsy were limited to the chest. A huge empyema was present in each pleural cavity, and fistulization into the chest wall had begun. Extension through the skin probably would have occurred within the next few days had the patient survived. The lungs each weighed 2400 grams. They were the seat of a very extensive purulent inflammatory process that involved every lobe of both lungs. In some areas consolidation was apparent, but in many other areas frank necrosis of pulmonary parenchyma had occurred, with the production of huge abscesses. Some of these had evacuated the exudate through large bronchi, with resultant cavitation. A large emphysematous bleb was present in the left apex. The remainder of the examination revealed no other organ involvement. Permission for examination of the head was refused, but involvement was not apparent in a portion of the lumbar spinal cord.

Microscopic examination revealed a simple purulent inflammation of the lungs entirely made up on neutrophils. No epithelioid cells, giant cells or other unusual features were noted. Extensive destruction of lung parenchyma, abscesses and cavity formation were obvious. Sections stained with hematoxylin and eosin revealed no etiologic agent. Acid-fast stains prepared in the usual manner showed no organisms. Gram's stains, however, showed huge numbers of gram-positive mycelial fragments characteristic of Nocardia (Fig. 3). Cultures of the lung revealed a pure growth of an aerobic mycelial fungus culturally and morphologically identical with *Nocardia asteroides*. The organism was pathogenic for guinea pigs. While it was not acid-fast when decolorized in the usual manner (with acid-alcohol), its acid-fast properties became apparent with more gentle decolorization (aqueous sufuric acid).

These mycelial fragments also were found in the sputum two days before death and were under study at the time the patient died. Cultures of the sputum obtained before death subsequently showed the same organism. All cultures and examinations for tubercle bacilli were negative. No other pyogenic organisms were demonstrated. Sensitivity studies showed this fungus to be resistant to erythromycin, penicillin and sulfadiazine; slight sensitivity was apparent to tetracycline and dihydrostreptomycin, while moderate sensitivity to chloramphenicol was noted.

Nocardiosis is an infection due to the fungus Nocardia sp. Although Nocardia is closely related morphologically and culturally to Actinomyces, it differs chiefly in that it is strictly aerobic and many species are acid-fast. Fragmentation of the finely branching mycelium into acid-fast rods frequently has resulted in an erroneous diagnosis of tuberculosis. The fungus is Gram-positive, cultures easily on standard media and can be separated from Actinomyces on the basis of its aerobic nature, acid-fast properties and pathogenicity

(Continued on Page 208)

Editorials

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

POST-SANATORIUM CARE OF TUBERCULOSIS

The markedly improved prognosis of the tuberculosis patient in the past decade has changed the attitude of the therapist in many ways. One danger is obvious. Undue optimism over present therapy can breed carelessness, neglect and eventual disappointment, either in the handling of an individual case or in a general program. Our new successes in tuberculosis therapy must not make us disregard careful and prolonged follow-up of every tuberculosis patient.

Post-sanatorium care starts with proper education of the patient while he is in the sanatorium, and is brought into sharp focus at the time of his discharge. To instruct the discharged patient to "see your family doctor regularly" and then close the sanatorium record was once fashionable though never medically wise. Today's patient should have on his discharge clear instructions regarding, and understanding of, the need for regular and faithful follow-up examinations to watch for return activity of his disease or tuberculosis complications. These instructions (a summary of which should not only be in the medical records but also available to his family physician and to the local Health Department) should include coverage of:

1. Daily schedule of rest and activity: Definite instructions should be given regarding the type and amount of daily activity permissible, including rehabilitation activities; the length of daily sleep and rest; possible need for recording of weight, temperature, respiratory or other complaints, or any other progress record the discharging physician may deem necessary. Such instructions should cover the interval until the first regular post-discharge examination (usually 6 to 12 weeks) and may be revised thereafter on each return.

2. Medication: If the patient is to continue on medication, directions regarding drug therapy must be clear and unequivocal as to dosage, duration of treatment, how and when to return for a drug refill, and the dangers both of neglect in taking medication regularly and of failure to report signs of drug reaction. To provide the patient with a printed form on which he is to record his actual drug intake day by day, for later review by his physician, may seem laborious but is well worthwhile.

3. Laboratory controls for the patient on drug therapy: None of the drugs used in tuberculosis therapy can be released to the outpatient with complete impunity or unconcern, for each can occasionally lead to serious reactions. Regular periodic blood counts (as for anemia due to para-aminosalicylic acid), liver function tests, and audiometric examinations (in long term streptomycin therapy), may detect early signs of damage and prevent serious developments. For the patient on well established therapy, post-sanatorium studies at three month intervals are generally sufficient.

4. Clinical, bacteriological and x-ray review: Such review should at first be frequent—say once in three months—with the interval gradually lengthened to one year as the patient's condition remains stable. A personal conference on each visit may be even more important than the routine chest film that is expected. The patient may by a question or two relieve himself of anxiety. The physician may detect signs of physical strain or of reactivation or complicating disease. His questions should relate not only to the chest but to any possible extrapulmonary foci as well. Thus a renal case may need cystoscopic re-evaluation or pyelography as well as or instead of chest x-ray.

Need for bacteriologic study must be carefully considered on each post-discharge examination. For the pulmonary case with expectoration, sputum microscopic *and* culture examinations at three month or longer intervals may suffice, but if sputum is scanty or non-obtainable, a series of three or more gastric specimens should be obtained periodically for culture or guinea pig study (microscopic study of gastric specimens is generally regarded as unreliable). Such series of gastric examinations are often advisable at three month intervals throughout the period of chemotherapy and again—as a test of effectiveness of therapy—at three months or even at six or twelve months after completion of the drug regimen. Again for the extrapulmonary case corresponding bacterial examinations of urine or other excretion or drainage material may be in order. And finally, any hint of reactivation of a tuberculous focus even years after apparent control, calls for intensive bacteriologic re-study.

How long should clinical and x-ray follow-up be maintained? The known tuberculosis case should be re-examined at least annually throughout his remaining lifetime, for tuberculosis can and does reactivate after many years of apparent inactivity.

5. Liaison with auxiliary medical services: The discharging physician should also establish close co-operation between the patient, the sanatorium out-patient department, and various official agencies. Thus, the local health department and public health nurse need advice regarding the patient's discharge status, medications, home care, return to school or work activities, and follow-up schedule. The State Division of Vocational Rehabilitation may need repeated guidance on training and job placement for those patients requiring a change of work. And where family incompatibility, financial stringency, or emotional conflicts make personal adjustment critical, social service and welfare agencies may provide invaluable help.

As already implied, good post-sanatorium care of the tuberculous presupposes good indoctrination and training of the patient before his discharge. The well trained patient usually is a co-operative patient. He who leaves prematurely, with training broken and incomplete, is prone to become delinquent in follow-up, and so is more likely to become a public health menace if untraced.

For the tuberculosis patient discharged from the sanatorium following diagnostic study but with no present or recent activity demonstrated, clinical, x-ray and bacteriological follow-up may of course be modified accordingly—say routine annual recheck only.

One factor which unfortunately limits many post-sanatorium care programs is economic pressure. If both the community and the patient are limited economically, ideal follow-up becomes difficult indeed. Most communities in the United States today have the means to set up creditable programs, including free care for the economically straightened patient, if determined to do so. Certainly no area in Minnesota needs to be satisfied with inadequate tuberculosis follow-up.

R. W. BACKUS, M.D.

MEDICAL LIBRARIANSHIP: PRODUCT OF A NOBLE TRADITION

The doctor approached the reference desk. Eyes of the Egyptian clerk were question marks as they looked up at him from the scroll of a current best seller Grecian myth. The doctor wanted all the literature available on alopecia. There was a jangle of keys, the slow forced action on an unused lock, a door opening on rusting hinges. In the vault-like space, several pottery jars could be seen. From one of these, a roll was selected, dust rubbed from the writing. It was handed to the waiting doctor, who took it eagerly. At the one table in the poorly lit room, he unrolled his treasure. This was it—the papyrus on medicine, later to become the famous Ebers papyrus. It described all that was known about the diseases of the time with almost one thousand recipes for their cure. More wonderful still, some human anatomy had been included! The time was 1500 B.C.

The doctor again approaches the reference desk. He speaks distinctly into a microphone. Air vibrations activate the mechanism of a giant vaculux in front of him. He wants all the material available on Majocchi's disease. A red light flashes. There is the purr of machinery set in motion. The doctor looks at his watch. The light changes to green. Rolls of film strips, discs and microcards, done up in neat packets begin emerging from the pneumatic delivery tube. He inserts an attached card into a slot that automatically charges the materials and operates the contact which releases them to him. In a near-by sound proof, air-conditioned carrel, micro-readers are ready, not only to amplify the print, but to reproduce at the push of a lever any portion of the journals that he wishes to take with him. He sighs with satisfaction and again looks at his watch. The whole process had taken less than fifteen seconds. This is 2050 A.D.

Between the ages of papyri and of automation, the medical library evolved from the small collections of physicians to the multi-thousand volumes of books and journals which make up modern medical libraries as they are known today. What went into this evolution? Undoubtedly, in the beginning, the painstaking copying by hand of papyri and manuscripts preserved medical treasure for posterity. Early manuscripts were always compilations of much older works, which suggests the great antiquity of interest in medical writing. As soon as printing was invented in 1450, medical literature literally tumbled from the presses. As early as 1686 a complaint appeared in the literature that "too many books are printed confusing and burdening the mind more than they instruct it." If the author of this remark lived now he could find listed in the latest edition of *World Medical Periodicals* 4,000 titles in medicine not including journals in fields related to medicine or from five to six thousand more periodicals containing scattered articles on the subject.

Understandably, physicians, themselves, may be considered the founders of both medical libraries and the profession of medical librarianship. Who better than they could appreciate the real values of healing buried in the scrolls and manuscripts of antiquity? And since, from earliest times, they represented the learned classes of society, it is logical that they were the ones to collect, interpret and preserve the books through which much of our culture is transmitted.

At first, the home or office of the physician was his library. As medical libraries became common in institutions, many physicians limited their library to a practical working collection and some, as a hobby or avocation, turned to the collections of rarities in the literature or to the searching out of papers and books in some highly specialized angle of medical learning. It can be stated with truth that there will never be an age in which physicians will not be leaders in their devotion to literature. Whether this love for books is natural or acquired, they are irrevocably committed to it. The fast moving pace of medical science has seen to this.

In addition to being the first to see the potentials in a collection of medical books, doctors have gone much further in their contributions to the medical library. Their far-sightedness early made them realize that if one physician's library was good, several combined ones would be better. And so they pooled their resources and formed society and institution libraries. Many hospitals owe their core collections to altruistic-minded staff members. Medical societies all over the world trace their origin to the same source.

In 1898, four American physicians met with four librarians and established what is known today as the Medical Library Association, one of the outstanding organizations in the current library world. The need for this enterprise was noted in an article written by C. D. Spivak in the *Philadelphia Medical Journal,* October, 1898, on "Medical Libraries of the United States." The author's statement, "There is no profession with so large and voluminous a literature and none in which its care is so neglected," summarized the thinking which gave birth to this project whose primary purpose was, and is, to encourage the formation of medical libraries.

Medical libraries in their cradle days were nurtured by the personal care of physicans. When the field became complex, physicians trained their secretaries to carry on while they retained the title of physician-librarian. In the meantime, the education of librarians was developing into a well defined science. In the first quarter of the twentieth century, physicians could begin to relinquish their work which they did so long and so well into the keeping of a profession descended from theirs and ancillary to them.

To Dr. John Shaw Billings, Surgeon General during the Civil War and later head librarian at the Library of the Surgeon General's Office, the forerunner of the National Library of Medicine in Washington, belongs the honor of the greatest contribution made by a single physician to the field of medical librarianship. His genius and foresight created one of the great reference tools for all times and is recognized as one of America's greatest contributions to medical science. This work, *The Index-Catalogue of the Library of the Surgeon-General's Office,* marked a goal in organized medical literature and was the forerunner of other great indexes, notably the *Index Medicus, The Quarterly Cumulative Index Medicus* and the *Current List of Medical Literature.*

Nor are doctors today less active or interested in the welfare of their medical libraries. Through membership dues, gifts and endowments, libraries, which are traditionally short of funds, are maintained according to high standards set up by the Medical Library Association and staffed by a growing number of qualified medical librarians.

Medical librarians, then, have a proud heritage. To their "founding fathers" can be traced ideals of dedication, of zeal for the furtherance and preservation of knowledge and a challenge of providing this, and future generations of physicians, with services abreast and in advance of the rapidly changing patterns in medical education and in research.

Sister Teresa Louise, C.S.J.
Medical Librarian

TRENDS IN MEDICINE

As our medical antennae have become ever more sensitive to the causes of illness and accidents, we are aware that scarcely an illness lacks its emotional component. We have become correspondingly aware that more and more "organic illnesses" have roots in emotional causes. The "bottled-up" hypertensive, the ulcer patient who "swallows his anger," the attention getting asthmatic or arthritic, or the rejecting and rejected colitis problem are examples of emotionally caused illness. We are not yet willing to attribute all cause to emotions, for powerful still are bacteria, metabolic abnormalities, and new growths. More and more however we accept the mind and spirit as powerful etiologic agents in illness.

Stress is a popular word and a good one. It is stress of work, ambition, fear, and insecurity which we believe causes ulcers or colitis. It is the stress of self-containment and fear which may cause hypertension. Possibly it is the stress of conflict between ambition and accomplishment, between creative talent and expression, which causes arthritis, dermatitis, and allergies. Though there are many still unwilling to accept completely the idea that stress may be the principal cause of an illness, yet most physicians agree that it is a large and vital factor.

We should not eliminate stress from our lives even were it possible to do so, for stress is a necessary spur to achievement and essential to accomplishment. There is stress which is productive, and that which is futile. Stress may become distress. When accomplishment decreases under stress, when physical symptoms appear because of it, then we must decrease the load or run the risk of organic illness.

All of us know individuals whose days are crammed with excitement, problems, decisions, with haste and hurry, uncertainty and tension. Yet they seem serene, and they remain robust and well, carrying these burdens indefinitely. Stress is the ingredient in their lives which makes such activity possible and keeps them productive to the point of genius, but stress is not distressing. Others of our acquaintance buckle at the smallest decisions and are exhausted by the mildest deviation from routine. Major decisions and emotions are impossible for them. Distress is ailing them, and eventually will obtrude as an organic lesion susceptible to pathologists' classification.

The rigid and closed mind is also a product of stress. The insecurity of aloneness, the fear of non-conformity, dislike of the unknown are part causes of the rigid mind. That individual whose stand on any problem is completely predictable may be a great and consistent thinker, but more often he is one whose pattern is set and who fears because of distress to change it. We all know individuals who are young at eighty, and others who are aged at forty. Of course cerebral arteriosclerosis is often a major factor in this premature aging, but emotional approach to life is equally important. Faith, confidence, self-appraisal, and courage are the ingredients from which the Greeks propounded "Know thyself" as a guiding motto for life.

Faith is the factor least measurable in man's total health, yet perhaps most crucial. Faith has literally moved mountains, and there are thousands of patients to testify to the curative power of true faith. Man must have faith to live a full life and without faith life becomes meaningless. No physician has not witnessed the power of faith, and no physician can afford to ignore this tremendous aid in his therapy. We all want and need faith; it is part of the whole man, but often it must be learned.

Do I imply that the examining doctor should hold a religious catechism in his periodic examination? Certainly not. But neither do I believe that faith can be ignored as "belonging only to the church." Knowledge of an individual's faith or absence thereof is often of great help in care, and faith must neither be touted nor scorned by the physician.

CHARLES S. HOUSTON, M.D.
Aspen, Colorado

Second in a series of editorials on Trends in Medicine.

RESILIENT FLOORING IN THE DOCTOR'S OFFICE

For the doctor's office needing a "face-lifting," resilient flooring can make an important contribution, since the floor is such a conspicuous area in any decoration scheme.

Every kind of resilient flooring—linoleum and plastics in sheet form and the tile family of asphalt, vinyl-asbestos, rubber, cork, plastic, and special types—offers many, many patterns to fit any taste or decorative theme. The installation of a new floor may be the only major change needed to transform an unrevamped office into a more attractive, more inviting space.

Besides decorative appeal, resilient flooring has other practical qualities that recommend it for use in doctors' offices. For one thing, it can be installed in hours—overnight, even—permitting renovation without the loss of a single working hour. Once the floor is installed, it's easy to keep clean and sanitary. Dirt can't stick to its smooth, lustrous surface, and a broom whisks it clean easily. Sweeping is the only care required daily, and an occasional scrubbing and waxing will help keep the floor like new.

The variety of colors and styles available with modern types of flooring is almost unlimited. Combinations of patterns can be used to add special effects to the basic design. For example, custom-designed insets can be laid into the floor in the form of "traffic lanes," a caduceus, or the name of a clinic.

Resilient flooring is quiet underfoot, for it cushions footsteps and reduces other noise that results from impact. This same quality of resilience makes the floor comfortable to walk upon. But for all its beauty, quiet and comfort, resilient flooring is durable and permanent when cemented properly to the subfloor. With the right sort of maintenance, it will give years of service without any appreciable loss of color and pattern design.

Before choosing a resilient floor for his office, the doctor should become acquainted with the types of flooring available.

Resilient flooring in sheet form offers the advantage of floors that are practically "seamless." There are two basic types.

Linoleum is probably the best-known and most widely used of all resilient flooring products, in both homes and commercial-institutional installations. With its coloring and patterns extending through to the backing, Linoleum is a long-wearing floor. It is grease-resistant and characterized by its smooth surface and even texture.

Plastic sheet flooring is one of the newest products in the flooring line. Ideal for highlighting modern, colorful decorative themes, it is made in a number of stylings. Quality plastic floors are resistant to abrasion and wear and will withstand the harmful effects of hot soapy water, alcohol, alkalis, dilute acids and most solvents.

Resilient tiles are being specified more than ever before. The ease of handling and replacement, coupled with unlimited design possibilities, has accounted largely for this increase in popularity.

Asphalt Tile, lowest in cost of all resilient floorings, is installable anywhere and is virtually unlimited in application. It is extremely durable, but is not quite so easy to maintain as most other types of quality resilient floors.

Vinyl-Asbestos Tile is fire-retardant and, like Asphalt Tile, can be installed even below grade-level. It is noted for the clear brilliance of its colors and may be safely used in areas subject to occasional spillage of acids.

Rubber Tile is a durable flooring with high resistance to wear and indentation. Its ability to cushion footsteps makes Rubber Tile particularly suitable where quiet is important.

Plastic Tile is a quality material recommended for all interiors where it is desirable to have the flooring match the architectural beauty of the room. It is highly stain-resistant, especially easy to keep clean, and installable in any location.

Cork Tile is most resilient—therefore quietest and most comfortable—of all floorings. It provides a distinctive appearance in areas where beauty and dignity are desired. However, it is not recommended for areas where excessive dirt and heavy foot traffic are likely to be encountered.

Custom Vinyl Cork Tile is a brand-new type of flooring that consists of Cork Tile with a layer of pure, clear vinyl plastic fused on as a wearing surface. Thus, it combines the beauty of natural cork with the durability and easy care requirements of plastics.

Linotile is especially processed for resistance to indentation and abrasion. It should not be confused with Linoleum cut into blocks. Tough and sturdy, Linotile is highly recommended for foyers and other "heavy-traffic" areas.

Linoleum Tile is Linoleum cut into squares to facilitate handling and installation. It is available in many of the same patterns as Linoleum sheet goods.

When selecting a resilient floor, it is well to remember that there is a right material available for every need. The doctor should study his own requirements, then consult a flooring dealer for help in picking the material that will give the most satisfactory service in the long run. Most flooring dealers and manufacturers have special facilities for assisting customers in choosing color schemes and designs.

C. Eugene Moore
Armstrong Cork Company

LIFE INSURANCE IN ESTATE PLANNING TODAY

Do You Have a Plan?

The young physician starting his practice should take the first steps to insure against two of the hazards that are common to everyone and concern us at every age level—*emergencies* and *premature death.*

A reserve fund for emergencies can be underwritten through disability income insurance or through a fund set aside from earnings or a combination of both.

Considering the hazard of premature death, Dr. B. L. Riese, addressing a recent graduating class of Northwestern University School of Medicine, told the young physicians that his classmates range in age from sixty-six to seventy-five years, that 67 per cent continue in active practice, 7 per cent are disabled, and that less than 30 per cent have been able to retire.

"If we may make a suggestion," Dr. Riese said, "to the young man starting out in practice, a suggestion born of long and bitter experience, it would be to buy all the life insurance they can afford to carry, in a sound company, and out of their surplus to buy bonds and stocks of basic industries, leaving the gold mines to the gamblers."

An essential element of a sound insurance program is that it is tailored to meet the specific needs of the individual. The young physician will find his need for insurance protection is the greatest when his funds available to buy it are the least adequate. So his insurance program is always going to include several compromises. His immediate need is for the most protection for the least cost. Thus his compromise may be for purchasing 5 or 10 year term insurance which can be converted to permanent insurance without physical examination. Another compromise might call for the purchase of family income or family maintenance type of life insurance.

Do You Have a Will?

Having made a start on the first two phases of his estate plan, the young physician should then complete the third phase; he should make a will. It has been said that a will is the most important document a man will ever sign, unless it is his application for a marriage license.

There is no other way in which a person can be sure that his estate, no matter how small, will be distributed according to his wishes at his death.

Do You Have an Interest in a Partnership?

It is often desirable to provide in advance for sale of a partner's interest to the surviving partners after his death. To provide funds for this purpose, insurance may be taken out on the life of each partner and made payable to the survivors. Difficult tax problems have been involved in such arrangements in the past—and there are new answers in the 1954 law.

Who should pay the premiums? Formerly it was thought advisable to avoid having any partner pay premiums on the policy insuring his life in order to bar the possibility of estate tax on the proceeds. Thus, for example, partner A would take out a policy insuring partner B and pay the premiums. B would take out and pay for a policy insuring A. Difficulty was encountered where A and B were of different ages, since the younger partner would have to pay higher premiums.

But the 1954 tax law removed this problem. It provides that the estate tax on insurance proceeds is not determined by who pays the premiums but by who owns the policy at the insured's death. If it is considered desirable, A can now pay the premiums on the policy insuring his own life; B the premiums on the policy insuring his life.

Should existing policies be used in setting up such an arrangement? It was formerly considered inadvisable to use existing policies to finance these cross-purchase agreements because of the possibility that the partner holding the transferred policy would have to pay income tax on receipt of the proceeds.

The 1954 law has solved this problem, too. It specifically exempts from income tax the proceeds of policies transferred to a partner of the insured.

This exemption is of special importance where there are more than two partners. If, for example, A, B and C each hold policies insuring the lives of his co-partners and A dies, B and C will want to acquire the policies held by A on their lives. This can now be done without fear of income tax.

It may be desirable to have the policies taken out by the partnership, rather than by the individual partners. This can simplify the arrangement where a large partnership is involved.

Alan W. Giles
Northwestern Life Insurance Company

KUDOS FOR THE STAFF OF LIFE

Analyses of popular breads from thirteen countries of the world reveal that enriched bread made in the United States ranks high as the Staff of Life.*

In this study, carried out under the direction of Dr. Robert S. Harris, Head of the Department of Nutrition of the Department of Food Technology at the Massachusetts Institute of Technology, each bread sample was analyzed for moisture, fat, protein, carbohydrate, ash, calcium, phosphorus, iron, thiamine, riboflavin, niacin, pantothenic acid, folic acid, vitamin B_6, lysine, methionine, and tryptophan. The samples were ranked as a source of individual nutrients and the average rank taken as an indication of comparative nutritional value.

By this rating system a bread from Finland made from a combination of whole wheat and white flours plus a high level of nonfat dry milk placed first in nutritional value. American enriched bread ranked second.

The flour used to make our enriched bread is milled from a wheat higher in protein than that available in most countries. A typical formula for this bread includes, in addition to other ingredients, four pounds of nonfat dry milk and two and one-half pounds of compressed yeast for each hundred pounds of flour.

To this blend is added an enrichment wafer containing sufficient thiamine, riboflavin, niacin, and iron to permit a guaranteed level of these nutrients in the finished loaf. Minimum and maximum levels have been established by the Definitions and Standards of Identity for enriched bread promulgated by the Food and Drug Administration. These standards also limit the ingredients which may be used by the baker to known safe substances whose use does not conflict with the best interest of the consumer.

The high nutritional value of American bread as compared with the bread of the rest of the world must be credited to many groups: the Food and Nutrition Board of the National Research Council, the Council on Foods and Nutrition of the American Medical Association, the American Public Health Association, the Food and Drug Administration, and the milling and baking industries. It was through the efforts of these groups that the word "enriched" was officially endorsed at the National Nutrition Conference for Defense which met in Washington in 1941.

Unlike previous attempts to improve white bread's nutritional value, addition of the enriching ingredients did not change bread's sought-after flavor or character. It was this added value that made it possible for enriched bread to become a cornerstone of national health.

Although the wartime enrichment order was recalled after World War II, twenty-eight states have since passed enrichment laws of their own. Enrichment in the remaining states is a voluntary service of the baker.

American Institute of Baking

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*Included in this study were breads from: Australia, Austria, Cuba, Denmark, Finland, France, Germany, Italy, Norway, Philippines, Sweden, Switzerland, and the United States.

CLINICAL-PATHOLOGIC CONFERENCE

(Continued from Page 201)

for guinea pigs. While Actinomyces usually demonstrates susceptibility to penicillin, the sensitivity of Nocardia to antibiotics varies, usually being greatest to the sulfonamides.

The method of human infection is speculative at present. Clinically, the infection commonly is either a chronic subcutaneous abscess (mycetoma) or a deep-seated infection (pulmonary or cerebral, or both). Pulmonary nocardiosis may mimic the various forms of tuberculosis and, for previously mentioned reasons, the fungus infection may be misdiagnosed and treated as tuberculosis. It has been estimated that as many as 10 per cent of patients hospitalized in some tuberculosis sanatoria are actually suffering from nocardiosis. The fungus demonstrates a predilection for the central nervous system, with fatal cerebral abscesses reported in patients whose lungs revealed no detectable evidence of infection at necropsy. Detection and diagnosis depend on suspicion. Treatment is by chemotherapy or the use of antibiotics as indicated by sensitivity studies.

DR. HARRINGTON: In retrospect, it should be pointed out that the organism causing nocardiosis is one fungus that explains adequately all phases of this case, and it is unlikely that it was a secondary infection.

It is unfortunate that the brain could not have been examined, since a significant number of patients with nocardiosis have involvement of the central nervous system.

Initial signs of lymphosarcoma in children may include petechiae, pallor, ecchymoses, enlarged lymph nodes, splenomegaly, hepatomegaly, skin nodules, weakness or anorexia.

MINNESOTA BLUE CROSS-BLUE SHIELD

Both the amount Minnesota Blue Shield paid physicians during 1957 for care of participant subscribers, and the number of doctors' services for which Blue Shield provided benefits were over 22 per cent greater than during 1956. Since the number of participant subscribers increased approximately six per cent over the number of subscribers enrolled at the end of 1956, the percentage increase in payment and services is primarily the result of expanded Blue Shield coverage and greater utilization.

Blue Shield provided benefits of $8,473,555 during 1957 for physicians' services rendered participant subscribers. This figure is equal to average monthly payments of over $706,000, and is more than $1,500,000 greater than the total amount paid by Blue Shield for care of subscribers during 1956. Blue Shield provided benefits of $6,919,348 in 1956 for physicians' services to participant subscribers.

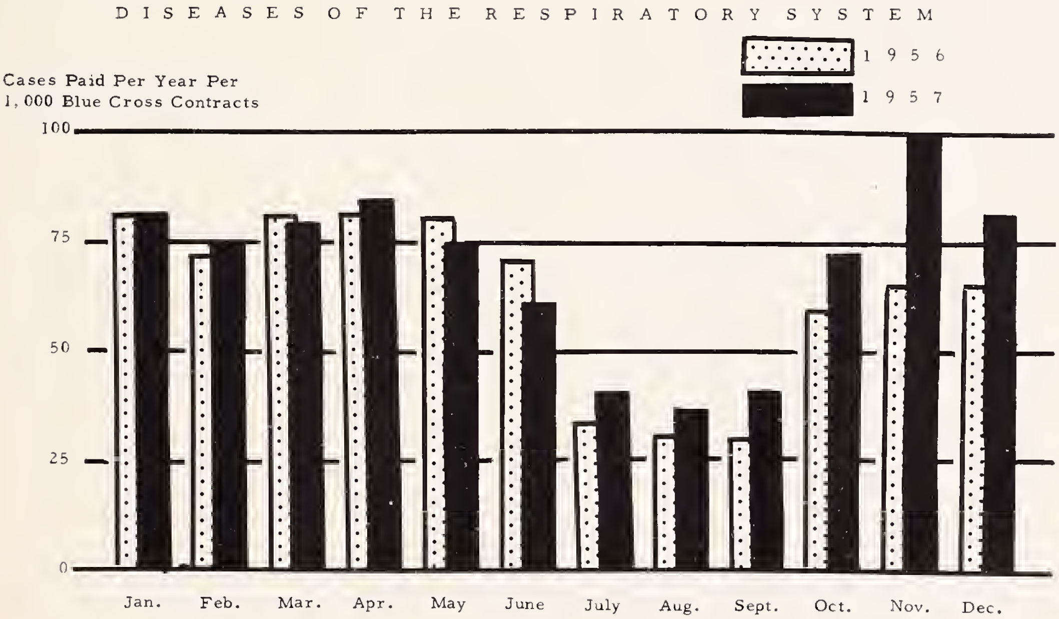
The number of physicians' services for which Blue Shield provided benefits during 1957 was 307,171, exceeding by more than 56,000 the number of doctors' services for which Blue Shield provided benefits during 1956.

Blue Shield participant subscribers numbered 869,985 as of December 31, 1957, or 46,883 participant subscribers more than the number enrolled as of the same date in 1956.

Payments in the form of hospital benefits for Minnesota Blue Cross participant subscribers reached an all time high of over $28,500,000 for the year ending December 31, 1957.

This all time high in benefits was provided to 202,704 participant Blue Cross subscribers for 1,233, 492 days of hospital care, reflecting an incidence rate of one case paid for every two Blue Cross contracts protected during 1957. This is a five per cent increase in frequency over 1956.

The increase in usage of Blue Cross benefits is attributable most importantly to a four per cent increase in the number of cases paid for conditions related to pregnancy, a six per cent increase in benefits paid for accident cases, and an eleven per cent increase in the number of patients hospitalized for diseases of the respiratory system.



Data collected by the Public Health Service indicates the peak in the recent Asian influenza outbreak was reached in the week ending November 2nd. The number of Blue Cross subscribers hospitalized for diseases of the respiratory system during the last quarter of the year 1957 follows the nationwide trend recorded for Asian influenza.

Respiratory cases took an abnormal upward trend during the last quarter of 1957 as compared to the previous year. The upward trend was largely due to an increase in the number of Blue Cross patients hospitalized for medical care such as influenza, pneumonia and upper respiratory infections. During the last quarter of 1957, 6,454 Blue Cross members and dependents received medical care in hospitals for respiratory diseases, compared to 3,636 during the same period of the preceding year.

Accident cases sent more Blue Cross subscribers to the hospital during 1957 than any illness or disease. Over 34,000 persons received Blue

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President's Letter

CIVIL DEFENSE AND CIVILIAN DISASTER

One Sunday afternoon, in 1939 or 1940, a tornado struck Anoka, Minnesota. There were a few people killed and quite a number injured. In a few minutes, a call went out over the radio for doctors to come to Anoka. Those who thought they could help started on their way. The rest of us did not happen to have the radio on. Half an hour or so later, the announcer asked that no more doctors come to the scene of the tornado; there were too many doctors and, besides, the roads were so filled with sightseers that the ambulances and service crews could hardly get into the town, and the disaster was not as bad as it seemed at first. In other words, planning was absent, the improvised arrangements were more or less hysterical and were finally interfered with by the morbidly curious who could only hinder what progress was being made.

Over the years since that time, we have slowly recognized that care of the injured in a small calamity requires forethought and planning just as it does in a disaster or catastrophe. However, as our population increases and the concentration of people becomes greater, the possibility of more injuries occurring simultaneously increases and the need for disaster planning becomes more urgent. Now, practically every hospital and almost every staff has an outline for procedure in case of a sudden influx of casualties. In fact, soon a disaster plan will be one of the requirements for hospital accreditation.

The usual pattern is selection of an area in the hospital as a receiving center, assignment of the staff to various teams, such as shock, transfusion, surgery, fractures, and psychiatric care. What is just as important is planning to bring in the laboratory staff for blood typing, transfusion, blood expanders, et cetera; the supply department for extra beds, blankets, and bedpans; and the kitchen and maintenance help simply to feed and house the whole group, staff as well as patients, and, equally important, enough administrative and clerical help so we know who is injured and where they are.

Now, if we enlarge the "ordinary" civilian disaster almost immeasurably, we can begin to conceive of the planning for civil defense. As Dr. Carl Waldron has done elsewhere in this issue, try to imagine an enormous disaster—a bomb which can not simply blow up a few buildings but which can wipe out the whole of Minneapolis and St. Paul with one blast. The bomb has a force of 20 megatons, in other words, the equivalent of 20 million tons of TNT. The Civil Defense planners, the Federal Civil Defense Administration, are working with the idea that such a bomb could be delivered but that there would be from two to six hours' warning. During that time, according to the present plan, the target areas would be evacuated. All roads would be made one way (out) and a speed of 30 miles an hour maintained, which makes such an operation imaginable. There would, then, be an enormous problem in caring for the healthy or the routinely sick, not to mention casualties. At first, the only concern was for care of the injured, and depots of supplies have been provided in northern Iowa and various parts of Minnesota to set up emergency treatment stations and emergency hospitals. The ordinary health care and sanitation for such a displaced population is now recognized as a really major problem, and when it comes, as we hope it never will, this may be the answer to the survival of the nation.

In the planning for such a major "national emergency" (an understatement, but the only words that seem to fit), it is recognized that the physicians of the target areas may be decimated and certainly will be displaced. Therefore, the burden of organized care will fall on the medical and paramedical professions in parts of the state away from the targets. The actual planning is being done by the physicians engaged full time in community care, the state and local health officers, and by such dedicated men as Dr. Carl Waldron of Minneapolis, who has spent almost full time in this field for about four years; Dr. Hans Schwyzer of St. Paul, who has given generously recently; and Drs. Edwin G. Benjamin and Nathan C. Plimpton, who have spent part time for twelve years and have a large experience in Minneapolis problems. Others have contributed greatly in St. Paul, Duluth and other areas around the state. However, it is recognized that the actual work will be done by physicians and others miles away from the cities.

The Federal Civil Defense Administration has made a contract with the American Medical Association for planning medical care in a national emergency. For various reasons, one of which is the relatively advanced planning in this state, the Twin City area has been selected for a pilot study. To make this survey valuable, the aid and co-operation of all the doctors is needed, especially those away from the cities. In the cities, the surviving doctors will simply be told where to go, but in the surrounding towns, the survey and plans will include not only medical abilities but people and supplies: nurses—active and inactive, dentists, veterinarians, people to carry stretchers, to set up beds, to cook, and to keep records. Supplies will be surveyed: supplies in doctors' offices, in hospitals, in drug stores, in wholesale houses and warehouses, even in hardware and furniture stores, besides the more obvious blood bank supplies.

In the care of the health of the "surviving population," the key person is the doctor in the area not hit by a bomb. The association of these key people is the State Medical Association. Therefore, the State Medical Association, through its president, urges your full co-operation and your earnest help. The Association office will do its best to keep you informed.

Because of the key position of the medical profession in care of the health of survivors and casualties, our part is being carefully planned. At the same time, we recognize that other people, other professions, are striving to increase understanding between nations, to prevent the bomb's ever being dropped. The best civil defense is the brotherhood of man and is the ideal toward which the world must grow. The role of medicine, however, until that ideal is attained, is to plan and to know the technique of survival in catastrophe, and to repair and to care for the people involved.

Horatio B Sweetser

President. Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

BEHAVIORAL SCIENCE NEEDS STRESSED

Powerful development of behavioral science—"the science of man"—to bolster national defense, secure the peace, and strengthen the United States in many ways, has been recommended by an informal study committee of fifteen persons.

The recommendations were made in a 7,000 word report which suggested establishment of an advisory panel of behavioral scientists to work with James Killian, Jr., science advisor to the president. The report also called for provision of greater public and private funds to stimulate research in the various sciences of man.

Chairman of the committee was Dr. James G. Miller, director, Mental Health Research Institute at the University of Michigan.

VA DOCTOR PAY HIKE ASKED

The Veterans Administration is asking congress for funds to grant an average 7 per cent increase to its doctors. The increasing difficulty of hiring and retaining physicians is the reason given for the request.

According to the VA, doctors in private practice make more money and even those on salaries in miscellaneous jobs at a greater advantage with their fringe benefits as well as fatter pay envelopes. Congress is expected to grant the request.

REORGANIZATION WOULD AFFECT GOVERNMENT MEDICAL CARE PROGRAM

Passage of a bill which would revolutionize the government's medical care mechanism is being urged by the Citizen's Committee for the Hover Report.

Observers feel that effects of the bill would be felt not only at the bureaucratic level but in the office of every physician who ever treats a Federal beneficiary or who possesses hospital privileges or who is subject to military obligation or who prescribes drugs—in short, all of them.

The bill, if adopted, would establish a Federal Advisory Council of Health, directly responsible to the President. Congress is expected to give the bill careful consideration before approving additional funds for hospital construction and doctor training programs.

WORKMEN'S COMPENSATION NEEDS OVERHAUL—MEANY

A special conference of labor representatives, April 15-17, has been called by George Meany, president of the AFL-CIO, to discuss overhaul of workmen's compensation laws.

Meany's statement announcing the conference said, in part,

> "The advance of medical knowledge concerning occupational diseases has not been reflected in the adaptation of state workmen's compensation laws to protect workers occupationally injured.
>
> On-the-job safety programs have been neglected and, with the exception of a few states, only token consideration, if any, has been provided for the physical, mental, and vocational rehabilitation of the injured worker."

AMA PROPOSES MODEL LAW TO CUT POISON DEATHS

A model law which would require precautionary labeling of hazardous substances in commercial, household, and industrial chemical products has been proposed by the AMA committee on toxology.

The proposed legislation was announced recently by Mr. Bernard E. Conley, Ph.D., secretary of the AMA committee, before a section of the New York Bar Association. Mr. Conley told the law group that the proposed legislation is intended as a model for uniform laws to require declaration of hazardous ingredients and warning statements on the label and in the accompanying literature of chemical products in the home and elsewhere.

Mr. Conley pointed out that the law is directed against those hazardous substances defined as toxic, irritating, sensitizing, corrosive, inflammable, explosive, or radioactive under customary or reasonably anticipated conditions of handling and use.

The law, as drafted by the AMA committee headed by Dr. Torald Sollmann, Cleveland, Ohio, would:

—Require the labeling of all chemical products containing hazardous substances which are not now regulated.

—Require the same labeling standards to apply to chemicals for export as those for domestic consumption, thereby obviating the common complaint that less-than-standard products are sold to foreign customers.

—Prohibit re-use of food and drug containers bearing their original labels.

—Require identification and warnings for strongly sensitizing chemicals which cause allergic or inflammatory reactions in living tissue on contact.

The committee's work had strong support not only from the chemical industry but also from the National Drug Trade Conference and the American Public Health Association.

QUACKERY CRACKDOWN IS ON THE MARCH IN ILLINOIS

The State of Illinois has recently taken a number of important steps to curtail the activities of quacks and unlicensed practitioners in the healing arts. Penalties for violators have been increased and examinations have been made more difficult. Provisions have been made for the publication and widespread distribution of an annual roster listing all licensed physicians. Also included in the publications will be up-to-date information on suspended and revoked licenses.

Members of the Minnesota State Medical Association are urged to be alert to possible influx of unqualified practitioners driven out of Illinois by the new regulations.

GP HEAD BLASTS COMPULSORY HEALTH INSURANCE

Politicians campaigning for compulsory health insurance might as well try to pass a law against Asian influenza. Dr. Malcom E. Phelps, El Reno, Oklahoma, head of the American Academy of General Practice stated recently:

"For more than twenty years," Dr. Phelps said, "an assortment of misguided politicians have tried to buy votes by offering 'free' medical care plans. They consistently fail to point out that medical care is like any other commodity; you get just what you pay for; no more.

"I don't care whether you call it socialized medicine or compulsory health insurance. There's no difference between the two. The most important consideration, as far as I'm concerned, is the health of my patients. They deserve the best medical care available and I don't want them misled by the wild promises of politicians who don't know the first thing about modern medical care.

"Any one who has talked to British doctors knows exactly what I mean, "Dr. Phelps added.

"In Britain, the government levies a fat tax and then tells the people that they are entitled to 'free' medical care. This is absurd. Under the British National Health Insurance plan, the doctor averages about four minutes per patient. The patient stops being a person and becomes a number on a government file folder, buried under a pile of complicated forms and health insurance regulations.

"Doctors have been accused of being against health and welfare plans and it's time someone set the record straight. We're not against anything that means better medical care. But we know, better than anyone in Washington, that you can't legislate good medical care any more than you can pass a law against Asian influenza.

"Some of the political pressure groups talk about a shortage of doctors. I'd like to remind them that the nation's medical schools have set eight new enrollment records in the last eight years. I'd also like to tell them that a great many doctors would retire rather than practice 'bargain basement' medicine under compulsory health insurance.

"If the American people want a shabby health care system, all they have to do is turn it over to professional politicians," Dr. Phelps concluded.

The American Academy of General Practice, with headquarters in Kansas City, Missouri, is the nation's second oldest medical association representing 23,000 family doctors.

SOCIAL SECURITY DISABILITY CHECKS NEAR $10 MILLION EACH MONTH

During the first six months of operation of its disability payments program, the Social Security Administration estimates that approximately 131,000 persons are now receiving payments. This total is expected to increase to nearly 200,000 by July of this year. The Social Security Administration points out that the average payments to the disabled are $72.24 a month. This compares with the national average of around $65 for all other retired.

This is explained in part by the fact that (a) the national average takes in many older persons who were not fully employed in previous years and (b) wages have gone up since the 1930s and early 40s. The disabled covered, on the other hand, come more recently from the labor market.

On the basis of 200,000 on the rolls next year and assuming the average monthly payment is unchanged (which is unlikely), the Disability Trust Fund will be paying out at the annual rate of nearly $175 million. SSA estimates that the fund had $525 million in it as of September. This is derived from the ¼ of 1 per cent payroll tax increase that went into effect January, 1957. Note: At the time of the hearings on the disability proposal, HEW Secretary Folsom pointed out that the program for the first full year would cost close to 200 million, rising to around $900 million in 1980.

"MD INTERNATIONAL" WELL RECEIVED IN MINNESOTA

The American Medical Association-Smith, Kline and French TV show, "MD International," was big news in the medical field recently.

Many at AMA headquarters who saw the one hour show over the NBC-TV network on Thursday evening, January 23, rated it the best of the March of Medicine series.

The *Chicago Tribune* critic said the fine show renewed his faith in the future of television.

"It contained scenes of such pathos, high purpose, and elemental humanity that it must be ranked high on the list of TV documentary films," the *Tribune* writer said. "Rave notices" also appeared in many other papers throughout the country.

This was the filmed story of American doctors in eight locations. The seven men crew made the 34,000 mile trek in 81 days—to Korea, Hong Kong, Borneo, Burma, Lebanon, Nepal, Ethiopia, and India. The film has a "cast of thousands"—and not one was an "extra."

The show was carried on 148 NBC network TV stations. In Minnesota "MD International" was aired over KSTP-TV, Twin Cities; KROC-TV, Rochester; and KDAL-TV, Duluth. Early estimates indicate that the presentation was viewed by 20 million persons.

Vice President Richard M. Nixon introduced the program.

The popular March of Medicine series has been a public service function of the AMA and Smith, Kline & French since 1952.

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(Continued from Page 191)

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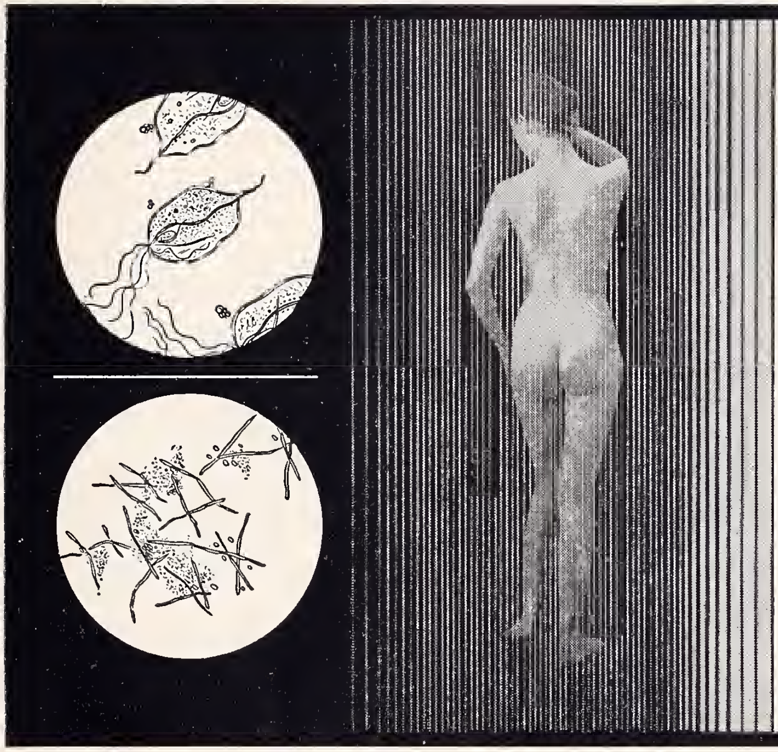

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SEARLE

In Memoriam

RICHARD B. HULLSIEK

Dr. Richard B. Hullsiek, chief medical officer of the regional veterans administration office at Fort Snelling, died February 2, 1957, of a heart attack, at the age of fifty-nine.

He had been in charge of the VA medical clinic at Fort Snelling since 1946. He lived on the post.

Dr. Hullsiek, a lifelong Twin Cities resident, attended Macalester College, St. Paul, and graduated from University of Minnesota Medical School in 1924.

A private practitioner in St. Paul until 1940, Dr. Hullsiek then entered the army as a medical officer. He served in Europe with the rank of colonel during World War II. He also was a veteran of World War I.

Dr. Hullsiek was a member of the American Medical Association, Minnesota State Medical Association, Ramsey County Medical Society and Medical Forum.

Survivors include his wife, Stella; a son, Richard, Jr., Bloomington; a daughter, Mrs. Donald P. Moffitt, Minneapolis, a brother, Dr. Harold E. Hullsiek, St. Paul, and a sister, Mrs. Henry Koll, Chicago.

JULIUS JOHNSON

Dr. Julius Johnson, Minneapolis physician for more than fifty years, died February 8, 1958. He was eighty years old. His specialty was Neurology and Psychiatry.

A native of Sacred Heart, Minnesota, he received his preliminary education in the rural schools of that community and at St. Cloud State Teachers College.

His medical education was obtained at the Minneapolis College of Physicians.

He interned for one year at St. Barnabas Hospital in Minneapolis. His post graduate work included four months at Harvard Medical School.

Dr. Johnson served on the staffs of Swedish, St. Barnabas, Fairview, St. Andrews, Deaconess and Glenwood hospitals.

He was a life member of the Minnesota State Medical Association, a member of Hennepin County Medical Society and the American Medical Association. Other memberships held by Dr. Johnson included: the American Psychiatric Association, Minnesota Society of Neurology and Psychiatry and Phi Rho Sigma medical fraternity.

Dr. Johnson also belonged to Wesley Methodist church, Masonic lodge 19, Scottish Rite and Zuhrah temple of the Shrine.

Survivors included his wife, Bess R., a daughter, Mrs. H. G. Creel, Palos Park, Illinois.

(Continued on Page A-48)

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(Continued from Page A-46)

RICHARD O. LEAVENWORTH

Dr. Richard O. Leavenworth, St. Paul, former chief of the eye, ear, nose and throat department at Ancker Hospital in St. Paul, and former clinical instructor at the University of Minnesota, died January 29, 1958. He was sixty-eight years old.

He was a graduate of the University of Minnesota. Dr. Leavenworth took post graduate work at a number of American and foreign universities.

Prior to fourteen months service in World War I in France, he practiced medicine in Glencoe, Minnesota. Following World War I, he returned to practice in St. Paul.

Dr. Leavenworth was a member of the staff's at Children's, St. Joseph's, St. Luke's and Miller Hospitals in St. Paul.

He belonged to Ramsey County, Minnesota State and American Medical Associations and was a fellow of Minnesota and American Academies of Ophthalmology and Otolaryngology.

A fellow also of American College of Surgeons, he was certified by the American Board of Ophthalmology.

Survivors are his wife, Geneva; two daughters, Mrs. Edmund A. Post, St. Paul, and Mrs. Harrison H. Cochran, Jr., San Gabriel, California; a son, Dr. Richard O., Jr., Excelsior, and a sister, Louise, Schenectady, New York.

CARL M. OBERG

Dr. Carl M. Oberg, Minneapolis, died February 6, 1958. He was eighty-one years of age. His specialty was eye, ear, nose and throat. He was born in Sweden in 1876.

Dr. Oberg was a graduate of the University of Minnesota Medical School, class of 1899.

He was a life member of the Minnesota State Medical Association, a member of the fifty year club, and the Hennepin County Medical Society.

He also belonged to the American Academy of Opthalmology and Otorhinolaryngology, Deaconess hospital medical and surgical society, Phi Rho Sigma medical fraternity, Swedish art institute, Swedish society, Anchor Masonic chapter and Zuhrah temple.

Survivors include a daughter, Margaret Oberg, Minneapolis.

LOREN J. O'CONNOR

Dr. Loren J. O'Connor, St. Paul physician, died December 14, 1957.

Born in Delano, Minnesota, he was fifty-six years old at the time of his death. He received his preliminary education at St. Thomas Academy and College in St. Paul.

His medical education was obtained at St. Louis University. He served one year of internship at Ancker Hospital in St. Paul. Dr. O'Connor held the following

(Continued on Page A-50)

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(Continued from Page A-48)

positions and affiliations: Associate Member of the Minnesota State Medical Association, a member of the Ramsey County Medical Society and the American Medical Association. He also served on the staffs of St. Joseph's, Bethesda and Midway Hospitals.

HARRIS RAYMOND SUTTON

Dr. Harris Raymond Sutton, sixty-nine, of Hoffman, died January 16, 1958, at the Douglas County Hospital in Alexandria. His death followed a two month illness caused by lung cancer.

A native of Prior Lake, Minnesota, he received his elementary education at Windom Institute at Montevideo. He took his A.B. degree at the University of Minnesota in 1912 and in 1916 he was awarded the Doctor of Medicine degree. He had his interne experience in Ancker Hospital, St. Paul, and from 1917 to 1920 he served in the United States Navy Medical Corps.

Dr. Sutton was a member of Nu Sigma Nu Medical Fraternity, Phoenix Lodge A.F. & A.M. No. 313 and the Hoffman Post of the American Legion. At the time of his death he was chairman of the board of education of Independent Consolidated School District No. 265, Hoffman, a post he had held for many years. He had also served several terms as village mayor and council member. He was a member of the Park Region District and County Medical Society, an active member of the Minnesota State Medical Association and the American Medical Association.

On May 31 last year Dr. Sutton's many years of medical practice and public service were recognized at a special Dr. Sutton Day observance at the Hoffman school auditorium. The event was under sponsorship of several Hoffman and Kensington organizations.

Survivors are his wife, Clara, two sons, Harley M. of Minneapolis and Forest of St. Paul; one daughter, Mrs. Richard (Shirley) Davis of LaCrosse, Wisconsin. He also leaves three grandchildren, Joni Gayne Sutton, Scott Davis and Richard Davis; two sisters, Maude L. Sutton of Batavia, Illinois, Cora S. Castle of San Francisco, California, and one brother, Leonard Sutton of St. Paul.

CLARENCE E. WILLCUTT

Dr. Clarence E. Willcutt died January 8, 1958, in Phoenix, Arizona. He was seventy-four years of age.

A former Minneapolis Obstetrician and Gynecologist, Dr. Willcutt had resided in Phoenix, Arizona, for a number of years because of ill health.

He was born in Ida County, Iowa. His medical training was obtained at the State University of Iowa.

Dr. Willcutt was an associate member of the Minnesota State Medical Association and a member of Hennepin County Medical Society.

Survivors include his wife, three sons, Robert of Minneapolis, Clarence of Minneapolis and Charles of Chicago; two sisters, Ethel Willcutt of Minneapolis, and Mrs. J. A. Gray of Iowa.

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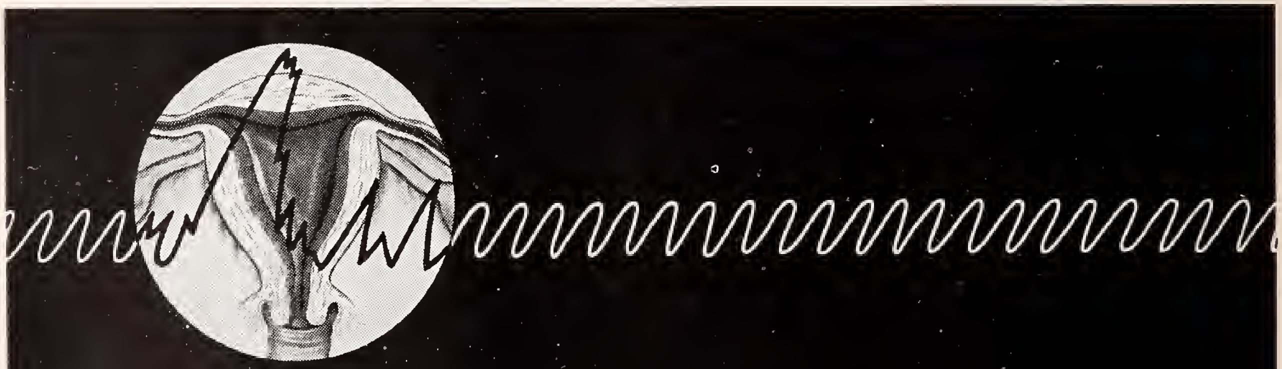

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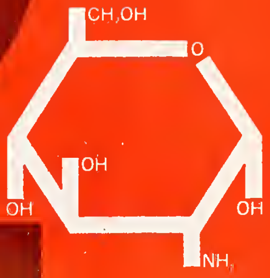


3

Achieved with the physiologic advantages of glucosamine, a normal human metabolite. Glucosamine, found widely in the body, is nontoxic and does not irritate the gastrointestinal tract; there is evidence that glucosamine may favorably influence the bacterial flora of the intestine. Further, it is sodium free and releases only four calories of energy per gram.

in new
well-tolerated

The most widely prescribed broad-spectrum antibiotic now potentiated with glucosamine, the enhancing agent of choice

COSA-TETRACYN*

GLUCOSAMINE-POTENTIATED TETRACYCLINE

Capsules, 250 mg., 125 mg.
Half strength (125 mg. capsules) for long-term indications or pediatric use.

Pfizer Pfizer Laboratories, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y.

*Trademark

Correspondence

KENNY INSTITUTE CLEFT PALATE CLINIC

Many months of planning and work by leaders in the various disciplines and agencies concerned with the care and treatment of patients suffering from cleft lip and/or cleft palate has culminated in the establishment and opening of a cleft palate diagnostic and evaluation clinic at the Elizabeth Kenny Institute, Minneapolis. This clinic, staffed by a rotating group of particularly qualified professional people representing the various disciplines involved in the long term management of persons having clefts, was made possible through a grant by the Kenny Foundation. Services of this group are available on an outpatient basis to all physicians seeing or treating cleft lip and/or cleft palate.

This clinic was predicated on the concept that the physical, mental and social potentials of persons suffering from cleft palates are best realized by a blending of all the professional disciplines that might be involved and that the over-all, long range program habilitation is most effectively carried out by the patient's own private physician and dentist. Thus, all referrals to this clinic must be made by the patient's physician. Recommendations and plans for therapy originating from this clinic will be forwarded to the referring physician as well as an invitation for the physician to participate in the formulation of these plans.

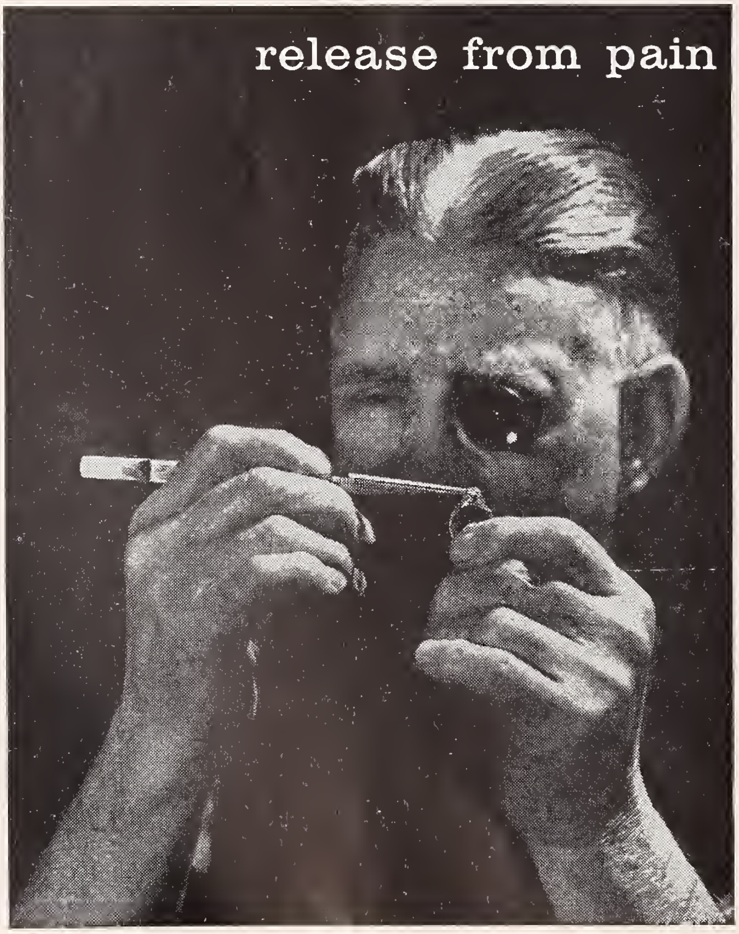
A complete study, including cephalograms, intra-oral radiographs, study models, intra-oral and extra-oral photographs, audiograms, speech analysis and a medical social service study will be made routinely. If desirable, further laboratory analysis, psychological evaluation and vocational guidance will be provided. The group organized to carry out this program includes a pediatrician, plastic surgeon, dentist, orthodontist, prosthodontist, psychologist, speech pathologist, medical social worker and, if necessary, an otolaryngologist and vocational counselor. Several complete panels have been organized to allow for a rotation of members throughout the year.

A complete analysis of the various studies made by each discipline involved will be formulated during a panel session into a long-range program of therapy. Referring physicians and other persons playing a role in the complete habilitation will be provided with all the available information. All persons involved in the care and treatment of the patient will be informed of the time the patient will be seen by the panel and invited to participate. All actual treatment will be conducted under the direction of the patient's own physician or dentist, not by members of the panel unless the panel member happens to be the patient's referring doctor.

Further information and referrals may be made by contacting the Kenny Institute Cleft Palate Clinic, Dr. Ralph B. Kersten, Coordinator, 1800 Chicago Avenue South, Minneapolis, Minnesota.

CERVICAL CARCINOMA

In a three-year study of cervical carcinoma, also at Parkland Memorial Hospital, Drs. M. A. Litton and P. O'B. Montgomery found evidence to support the assumption that social and environmental factors are important causes of cervical cancer. Early marriage, early childbirth and non-circumcision appeared to be significant factors in the patients studied.—*Dallas Medical Journal,* December, 1957.



release from pain and inflammation

with **BUFFERIN®**

IN ARTHRITIS

salicylate benefits with
minimal salicylate drawbacks

Rapid and prolonged relief — with less intolerance. The analgesic and specific anti-inflammatory action of BUFFERIN helps reduce pain and joint edema—comfortably. BUFFERIN caused no gastric distress in 70 per cent of hospitalized arthritics with proved intolerance to aspirin. (Arthritics are at least 3 to 10 times as intolerant to straight aspirin as the general population.[1])

No sodium accumulation. Because BUFFERIN is sodium free, massive dosage for prolonged periods will not cause sodium accumulation or edema, even in cardiovascular cases.

Each *sodium-free* BUFFERIN tablet contains acetylsalicylic acid, 5 grains, and the antacids magnesium carbonate and aluminum glycinate.

Reference: 1. J.A.M.A. 158:386 (June 4) 1955.

ANOTHER FINE PRODUCT OF BRISTOL-MYERS

Bristol-Myers Company
19 West 50 St., New York 20, N. Y

"Since we've had him on NEOHYDRIN he can walk without dyspnea. I wouldn't have believed it possible a month ago."

oral
organomercurial
diuretic

TABLET
NEOHYDRIN®
BRAND OF CHLORMERODRIN

LAKESIDE

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Meetings and Announcements

STATE

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

Minnesota Surgical Society, spring meeting, Rochester, Minnesota, May 9, 1958. O. H. Beahrs, M.D., Secretary, Rochester, Minnesota.

NATIONAL

Aero Medical Association, annual meeting, Statler Hotel, Washington, D. C., March 24-26, 1958.

American Congress of Physical Medicine and Rehabilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

National Health Forum, Sheraton Hotel, Philadelphia, Pennsylvania, March 18-20, 1958.

National Industrial Health Conference, 13th session, in Atlantic City, New Jersey, April 19-25, 1958.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

International Society of Internal Medicine, Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy Association, Paris, France, October 19-26, 1958.

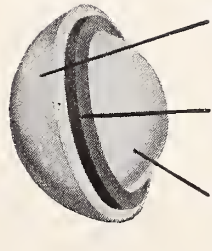
World Congress of Gastroenterology, Washington, D. C., May 25-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

(Continued on Page A-56)

NEW *"flavor-timed" dual-action*
CORONARY VASODILATOR

Dilcoron TRADEMARK

ORAL (tablet swallowed whole)
for dependable prophylaxis
SUBLINGUAL-ORAL
for immediate and sustained relief
of **ANGINA PECTORIS**



NITROGLYCERIN—
0.4 mg. (1/150 grain)—acts quickly
CITRUS "FLAVOR-TIMER"—
signals patient when to swallow
PENTAERYTHRITOL TETRANITRATE—
15 mg. (1/4 grain)—prolongs action

For continuing prophylaxis patient swallows the entire Dilcoron tablet.
Average prophylactic dose:
1 tablet four times daily.
Therapeutic dose:
1 tablet held under the tongue until citrus flavor disappears, then swallowed.

Bottles of 100.

Winthrop LABORATORIES NEW YORK 18, N. Y.

If
Monilial overgrowth
is a factor

Achrostatin* V

TETRACYCLINE (PHOSPHATE-BUFFERED) AND NYSTATIN

Combines ACHROMYCIN V with NYSTATIN

SUPPLIED:

CAPSULES contain 250 mg. tetracycline HCl equivalent (phosphate-buffered) and 250,000 units Nystatin. ORAL SUSPENSION (cherry-mint flavored) Each 5 cc. teaspoonful contains 125 mg. tetracycline HCl equivalent (phosphate-buffered) and 125,000 units Nystatin.

DOSAGE:

Basic oral dosage (6-7 mg. per lb. body weight per day) in the average adult is 4 capsules or 8 tsp. of ACHROSTATIN V per day, equivalent to 1 Gm. of ACHROMYCIN V.

ACHROSTATIN V combines ACHROMYCIN† V ...the new rapid-acting oral form of ACHROMYCIN† Tetracycline...noted for its outstanding effectiveness against more than 50 different infections ...and NYSTATIN...the antifungal specific. ACHROSTATIN V provides particularly effective therapy for those patients prone to monilial overgrowth during a protracted course of antibiotic treatment.

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, N. Y.
*Trademark †Reg. U. S. Pat. Off.

Lederle

(Continued from Page A-54)

POSTGRADUATE COURSES ANNOUNCED

New York University-Bellevue Medical Center's Post-Graduate Medical School offers the following postgraduate courses to be given or started during the month of May, 1958:

Symposium on Dermatology and Syphilology (for Dermatologists)—May 19 through 23

A full-time course of five days' duration. Given under the direction of Professor Marion B. Sulzberger. Maximum class 20. Tuition: $100.

Cardiology—May 5 through 23

A full-time, three weeks' comprehensive course. Given under the direction of Dr. Charles A. Poindexter. Maximum class 40. Tuition: $250.

Advanced Course in Urology (for Specialists)—May 12 through 23

A full-time course of two weeks' duration. Given under the direction of Professor Robert S. Hotchkiss. Tuition: $175.00.

OPHTHALMOLOGY FELLOWSHIP ESTABLISHED

The Department of Ophthalmology of the New York University-Bellevue Medical Center announces the establishment of the Daniel B. Kirby Fellowship for Research in Ophthalmology.

To be eligible for this award, the candidate must have had a thorough grounding in ophthalmology, roughly equivalent to a three-year residency, or should have acquired equivalent competence in a basic field related to ophthalmology.

Fellows will be expected to continue work on basic problems in such fields as ocular chemistry, physiology, pathology, or experimental surgery, and will be supplied laboratory space and facilities in the Center. The initial appointment will be for one year at a basic stipend of $7,000, and will be subject to renewal for two additional years.

SUMMER CAMP FOR DIABETIC CHILDREN

The summer camp for diabetic children will be conducted for the tenth year under the auspices of the Chicago Diabetes Association from July 20 through August 10, at Holiday Home, Lake Geneva, Wisconsin. Boys and girls from eight through fourteen years of age are eligible.

AURORA MEDICAL STUDENT WINS RURAL SCHOLARSHIP

Mr. Edward S. Peterka, of Aurora, has been awarded the 1957 Rural Medical Scholarship sponsored annually by the Minnesota State Medical Association.

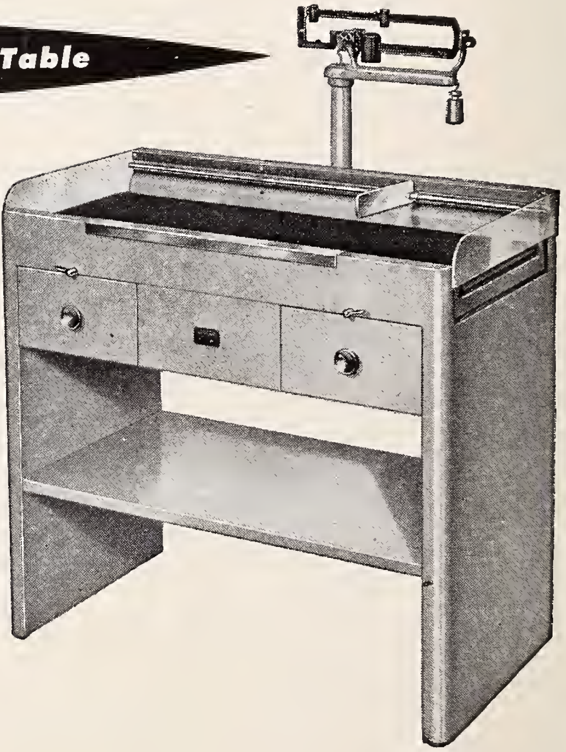
The recipient is a freshman in the University of Minnesota Medical School. He is a graduate of the Aurora Public Schools and attended Virginia Junior College prior to enrolling in the University of Minnesota.

In 1946, when he was a senior in high school, his

(Continued on Page A-58)

New STEELINE Pediatric Table

Eases the burden of the physician's daily work



Includes built-in tare balance scale, built-in measuring rod, foam rubber cushion and electrical outlets with cord and plug—there are no accessories to buy. Top quality construction features include all-welded steel body, two roomy drawers and a large open compartment with shelf, providing easy, unhampered access to instruments and supplies. Front edge is protected from wear by a stainless steel baffle plate. Despite the fact that this new table has been designed to provide the physician with the best modern equipment, efficient modern production methods in our own factory permit us to keep the price well below that of comparable models. Write today for complete information.

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STOP respiratory congestion orally

relief in minutes . . lasts for hours

In the common cold, nasal allergies, sinusitis, and postnasal drip, one timed-release Triaminic tablet brings welcome relief of symptoms *in minutes.* Running noses stop, clogged noses open—and *stay* open for 6 to 8 hours. The patient can breathe again.

With *topical* decongestants, "unfortunately, the period of decongestion is often followed by a phase of secondary reaction during which the congestion may be equal to, if not greater than, the original condition. . . ."* The patient then must reapply the medication and the vicious cycle is repeated, resulting in local overtreatment, pathological changes in nasal mucosa, and frequently "nose drop addiction."

Triaminic does not cause secondary congestion, eliminates local overtreatment and consequent nasal pathology.

*Morrison, L. F.: Arch. Otolaryng. 59:48-53 (Jan.) 1954.

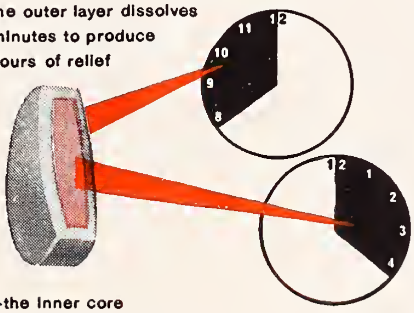
Each double-dose "timed-release" TRIAMINIC *Tablet contains:*

Phenylpropanolamine hydrochloride	50 mg.
Pyrilamine maleate	25 mg.
Pheniramine maleate	25 mg.

Dosage: 1 tablet in the morning, afternoon, and in the evening if needed.

Each double-dose "timed-release" tablet keeps nasal passages clear for 6 to 8 hours—provides "around-the-clock" freedom from congestion on just three tablets a day

first—the outer layer dissolves within minutes to produce 3 to 4 hours of relief



then—the inner core disintegrates to give 3 to 4 more hours of relief

Also available: Triaminic Syrup, for children and those adults who prefer a liquid medication.

Triaminic®

"timed-release" tablets

STOP *running noses . . . and open stuffed noses orally*

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska • Peterborough, Canada

For active mothers-to-be

Ulvical® (Ulmer) assures these modern ladies-in-waiting of a generous amount of iron and calcium plus the recognized prenatal vitamins . . . in a small tablet . . . especially designed for rapid assimilation and easy toleration.

For maximal utilization . . . timed release of these essentials is accomplished by special coatings controlled to dissolve at the pH of the stomach, duodenum and jejunum.

Important, too, calcium pyrophosphate contains larger amounts of elementary calcium which is quickly metabolized to increase plasma blood levels. Leg cramps are quickly relieved; small daily doses sustain this relief.

Each tablet contains:

Calcium Pyrophosphate (Ca 150 mg., P 120 mg.), 7½ gr.
Ferrous Sulfate USP (Fe 38 mg.) 3 gr. 3.8 M.D.R.
Vitamin A (Ester) 1500 USP Units ⅜ M.D.R.
Vitamin D (Irradiated Ergosterol) 200 USP Units ½ M.D.R.
Thiamine Mononitrate (B_1) 1 mg. 1 M.D.R.
Riboflavin (B_2) . . . . 2 mg. 2 M.D.R.
Ascorbic Acid (C) 16.66 mg. ½ M.D.R.
Vitamin E . . . . 2.2 I U.*
(from d alpha tocopheryl acetate N.F.)
M. D. R. — Minimum Daily Requirement. *M.D.R. not established.
Dose: One or two tablets three times a day.

Ulvical®

ULMER

MM—358b

THE ULMER PHARMACAL COMPANY
1400 HARMON • MINNEAPOLIS 3, MINNESOTA

(Continued from Page A-56)

father died. Following the death of his father, he spent the next two years working to support his widowed mother and a younger sister. In spite of his role as a breadwinner, he was able to save funds for his future medical education. His military experience includes two years with the United States Army in Korea.

The rural scholarship award was established six years ago by the Association to assist worthy and outstanding students, meet the high cost of medical education and to insure a supply of physicians in nonmetropolitan Minnesota communities.

Dr. Richard Engwall, the first person to receive this scholarship, is now a practicing physician in Ivanhoe, Minnesota. Other scholarship recipients, now attending the University of Minnesota Medical School include: Leland Christenson, Marshall; Myron Doebler, Princeton; Carl E. Christenson, Stanchfield; and Vincent R. Hunt, Anoka.

CRIPPLED CHILDREN SERVICES CLINIC SCHEDULE, SPRING OF 1958

St. Cloud—March 11-12—Bethlehem Lutheran Church—E. Stearns, Benton, Sherburne, So. Mille Lacs

Grand Rapids — March 12-13 — Court House — Itasca, Cass (Remer and Longville area)

Cloquet — March 19 — National Guard Armory — Pine and Carlton

Albert Lea—April 2—Freeborn

Brainerd — April 8 — First Methodist Church — Aitkin, Crow Wing, Cass, No. Todd, Wadena and No. Mille Lacs

Hibbing — April 10-11 — National Guard Armory — St. Louis, W. Range

Detroit Lakes—April 23—Armory—Clay, Becker, Mahnomen

Thief River Falls — April 29-30 — Trinity Lutheran Church—Kittson, Marshall, Pennington, Red Lake, Roseau

Gaylord—April 30—City Hall—Brown, Carver, Scott, McLeod, Nicollet, Sibley, LeSueur

Duluth—May 7—United Baptist Church—Lake, Cook, So. St. Louis

Fergus Falls—May 8—Armory—Ottertail, Wilkin

International Falls—May 16—Koochiching, Lake of the Woods

Faribault—May 21—St. Luke's Evangelical Church—Dakota, Goodhue, Rice, Steele

Worthington—May 24—Cottonwood, Jackson, Murray, Nobles, Pipestone, Rock

Morris—June 3—High School—Swift, Stevens

All new patients are requested to bring written referrals from their family physician.

All patients should have appointments arranged locally.

Note: Gillette State Hospital patients will not be attending any orthopedic field clinics.

(Continued on Page A-60)

FAST RELIEF... AND ON THE JOB

IN ALL DIARRHEAS . . . REGARDLESS OF ETIOLOGY

comprehensive control with **CREMOMYCIN**®

SULFASUXIDINE*® PECTIN-KAOLIN-NEOMYCIN SUSPENSION

SOOTHING ACTION . . . Kaolin and pectin coat and soothe the inflamed mucosa, adsorb toxins and help reduce intestinal hypermotility.

BROAD THERAPY . . . The combined antibacterial effectiveness of neomycin and Sulfasuxidine is concentrated in the bowel since the absorption of both agents is negligible.

LOCAL IRRITATION IS REDUCED and control is instituted against spread of infective organisms and loss of body fluid.

PALATABLE creamy pink, fruit-flavored CREMOMYCIN is pleasant tasting, readily accepted by patients of all ages.

*Sulfasuxidine is a trade-mark of Merck & Co., Inc.

MSD **MERCK SHARP & DOHME**
DIVISION OF MERCK & CO., Inc., PHILADELPHIA 1, PA.

a new white,
super absorbent
TREATMENT TOWEL

- Lint free
- Sanitary
- Pure White
- Economical

These treatment towels are not like ordinary towels because they are of three-ply construction and Melamine plastic treated for wet strength. In addition, they are super absorbent, economical, lint free and sanitary. They can be autoclaved and used for sterile drapes. 14x18¼ inches in size . . . pure snowy white and packed 500 to the case.

Write for more information and prices. MM-358a

Physicians & Hospitals Supply Co.

1400 Harmon Place • Minneapolis 3, Minn.

(Continued from Page A-58)

MINNESOTA OBSTETRICAL AND GYNECOLOGICAL SOCIETY

The Minnesota Obstetrical and Gynecological Society will meet on May 3, 1958, in Rochester, Minnesota. Clinical sessions will be held in Mann Hall; surgical clinics, at St. Mary's and Methodist Hospitals.

CONTINUATION COURSES

Medical continuation courses to be presented at the Center for Continuation Study, University of Minnesota are as follows:

March 20-22—Surgery for Surgeons.
March 31-April 2—Gynecology for Specialists.
April 7-9—Radiology for General Physicians.
April 10-12—Arthritis and Physical Medicine for General Physicians.
April 12—Trauma for General Physicians.
April 14-16—Gastroenterology for General Physicians.
May 5-9—Electrocardiography for General Physicians and Specialists.
May 12-16—Proctology for General Physicians.

For further information concerning the above courses, write to the Director, Department of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.

MINNESOTA BLUE CROSS-BLUE SHIELD

(Continued from Page 209)

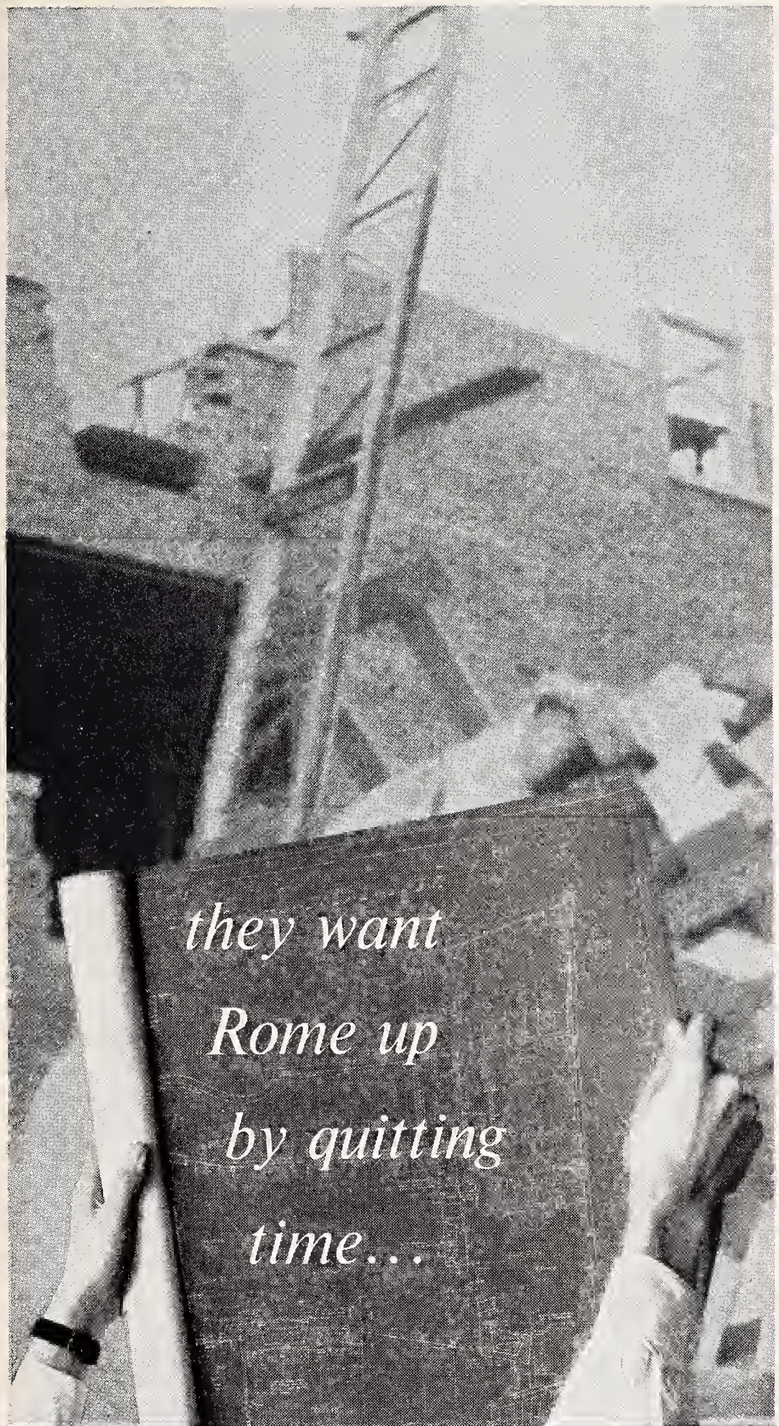
Cross benefits for the treatment of accidental injuries and poisonings.

Conditions relating to pregnancy ranked second in frequency of occurrence among Blue Cross subscribers during the year. Approximately 32,500 patients received Blue Cross benefits totaling over $4,200,000 for confinements and conditions related to pregnancy. During the year, 26,808 Blue Cross babies were born, compared to 24,165 in 1956.

Ranking third in reasons for hospitalization of Blue Cross subscribers, was respiratory illness. As noted, the last quarter upswing in this category resulted in the over-all high incidence rate for total Blue Cross benefits paid to subscribers.

Diseases of the digestive system ranked fourth in the leading reasons for incurred hospital cases during 1957, with approximately 25,000 Blue Cross subscribers being admitted to the hospital. Blue Cross benefits for these cases were higher than in any other category, accounting for approximately $4,800,000 of hospital expense, or 17 per cent of total Blue Cross hospital expense incurred during the year.

Blue Cross membership continued to grow during 1957. As of December 31, 1957, there was a total of 431,337 Blue Cross contracts in effect, representing 1,137,902 participant subscribers. This was a three per cent gain over 1956.



To cut daytime lethargy

(and keep rauwolfia potency)

in treatment of hypertension:

Mounting clinical evidence confirms the view that HARMONYL produces much less lethargy while reducing blood pressure effectively. In the most recent study[1], HARMONYL was evaluated in comparison with reserpine and other rauwolfia alkaloids. HARMONYL *was the only alkaloid which produced a hypotensive response closely matching that of reserpine, coupled with a greatly reduced rate of lethargy.* Only one HARMONYL patient in 20 showed lethargy, while an average of 11 out of 20 showed lethargy with reserpine, and 10 out of 20 with the alseroxylon fraction.

Abbott

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for your hypertensives who must stay on the job

Harmonyl*

(DESERPIDINE, ABBOTT)

while the drug works effectively . . . so does the patient

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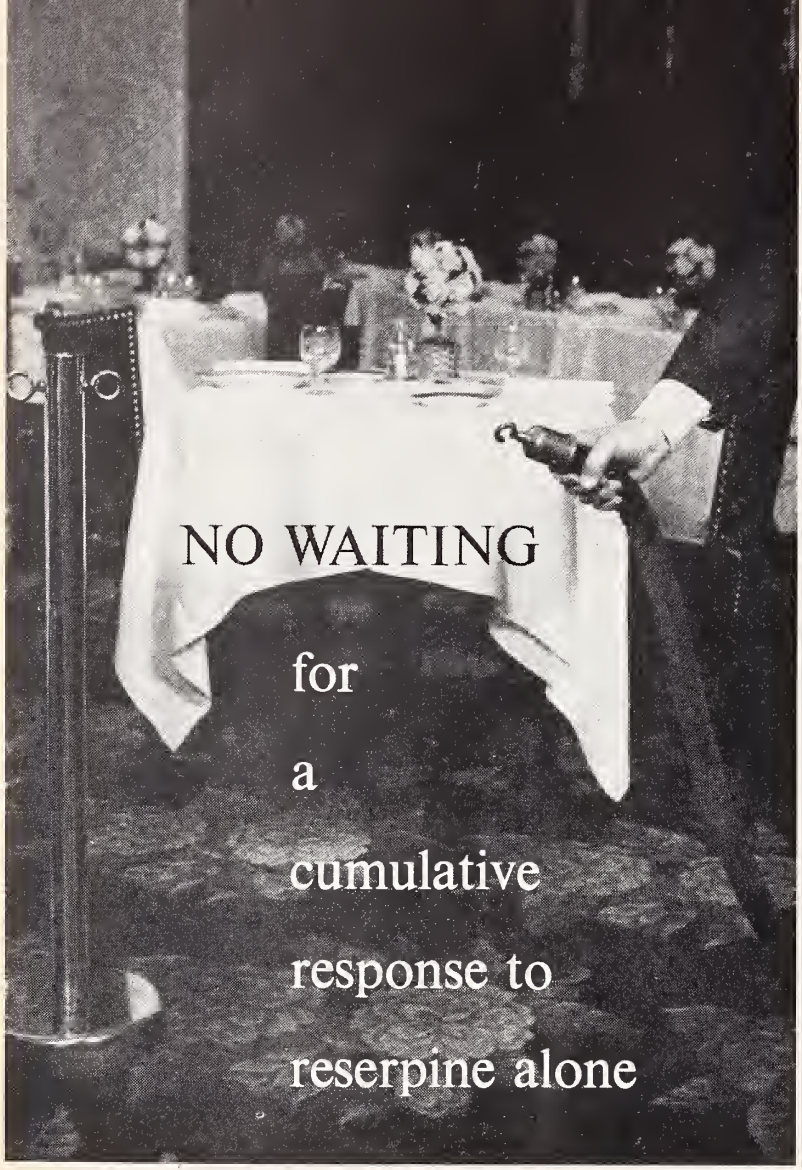
[1] Comparative Effects of Various Rauwolfia Alkaloids in Hypertension; *Diseases of the Chest;* in press.

*TRADEMARK



in anxiety and hypertension
NEW *fast-acting*

filmtab® **Harmonyl-N***
(Harmonyl* and Nembutal®)

Calmer days, more restful nights starting *first* day of treatment, through synergistic action of HARMONYL (Deserpidine, Abbott) and NEMBUTAL (Pentobarbital, Abbott). Lower therapeutic doses, lower incidence of side effects. Each HARMONYL-N Filmtab contains 30 mg. NEMBUTAL Calcium and 0.25 mg. HARMONYL. Each HARMONYL-N *Half-Strength* Filmtab combines 15 mg. NEMBUTAL Calcium and 0.1 mg. HARMONYL. Abbott

®Filmtab—Film-sealed tablets, Abbott; pat. applied for
801060 *Trademark

Woman's Auxiliary

WHO NEWS HIGHLIGHTS MID-WINTER BOARD MEETING

The Mid-Winter Board Meeting of the Woman's Auxiliary was held Wednesday, February 5, at the Minnesota Club in St. Paul. Mrs. C. L. Oppegaard presided over the meeting.

The business agenda for the day included a special legislative report by Mrs. L. Raymond Scherer. The possibility of raising dues was also discussed.

Highlight of the meeting was the presentation of a progress report of World Health Organization activities by Dr. Robert N. Barr.

Dr. Barr explained that the tenth anniversary meeting of the World Health Organization will be held in Minneapolis, May 26 to June 14, immediately following the annual auxiliary meeting. The first two days of the observance will be devoted to a tenth anniversary celebration in the Minneapolis Auditorium. An invitation has been extended to President Eisenhower to attend the celebration on May 26.

Dedication of the new Minneapolis Health Center will be held prior to the opening sessions of WHO.

Plenary sessions will begin May 28 and will be held several times each week in the Minneapolis Vocational High School Auditorium. They will break up into individual meetings which will be held at the Leamington and Curtis Hotels in Minneapolis.

Tentative plans include an official reception for WHO delegates at the State Capitol in St. Paul.

Dr. Barr estimated that as many as 22,000 persons representing 88 nations might attend the WHO Assembly, the first ever to be held in the United States.

In addition to the 400 official delegates, there will be the WHO staff from Geneva, representatives of many governmental departments, voluntary health and welfare agencies, and the health workers throughout the nation.

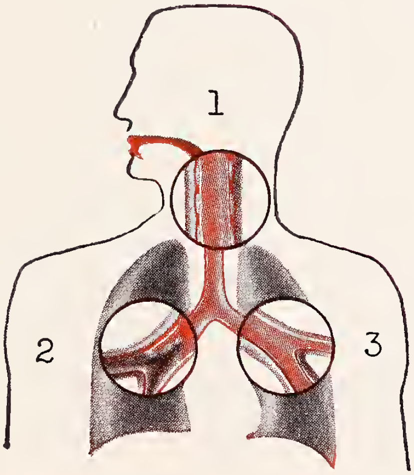
According to Dr. Barr, Minnesota will be the center of world health throughout the assembly. The importance of WHO to peace and stability is recognized by leaders the world over.

Dr. Barr referred to Senators Edward Thye and Hubert Humphrey and to Governor Orville Freeman for their joint untiring efforts to have Minnesota chosen the site of the first United States Assembly of WHO during Minnesota's Centennial Celebration.

The eleventh annual assembly of the World Health Organization in Minneapolis will provide members of the auxiliary an excellent opportunity to actively participate in this observance. Dr. Barr pointed out that plans are to have members of the auxiliary included on the hospitality committees which would be charged with the responsibility of arranging social events in twin city homes for delegates and their wives, during the first weekend of WHO. The following weekend the group will work all day Saturday in order to tour the

(Continued on Page A-64)

breaks up cough*



*Drawing shows how 3-pronged attack of Pyribenzamine Expectorant with Ephedrine breaks up cough by: (1) reducing histamine-induced congestion and irritation throughout the respiratory tract; (2) liquefying thick and tenacious mucus; (3) relaxing bronchioles. Pyribenzamine Expectorant with Codeine and Ephedrine also available (exempt narcotic). Pyribenzamine® citrate (tripelennamine citrate CIBA). **C I B A**

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Assure

MINIMAL DOSAGE RADIOGRAPHY

REDUCE

patient-dosage by

25%

and still get same quality radiographs

Switch to PIX
new MDR film processing solutions by
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SERVICE BROWN & DAY INC. SAINT PAUL U.S.A. QUALITY

62-64 EAST 5TH ST.
St. Paul 1, Minn.

(Continued from Page A-62)

Mayo Clinic on Monday. On the way to Rochester they will visit farms and meet rural Minnesotans. Sightseeing tours of industrial, cultural, medical and educational centers in the twin city area are also planned.

In addition to acting as hostesses for the visitors, the members of the auxiliary were also asked to consider fund-raising methods which would help finance the project.

WOMEN HEMOPHILIC?

For years it was taught that no woman is ever hemophilic, but well-authenticated cases of hemophilia in women are now on record. The disease can appear in a woman when a hemophilic man lives long enough to marry and have children by a woman who is a carrier. Today, a great effort is being made to secure adequate amounts of the antihemophilic globulin which may prevent hemophiliacs from dying of hemorrhages. Such a substance recently has become available.—Walter C. Alvarez, *Modern Medicine*, April 15, 1957.

Wilms' tumors comprise 20 per cent of all childhood cancer and 54 per cent appear before the age of three.

* * *

"Pot belly" is a classical sign of Wilms' tumor in children.

* * *

The sooner a patient with Wilms' tumor is diagnosed, the greater the chance for cure.

A MODERN PRIVATE SANITARIUM

for the Diagnosis, Care and Treatment of Nervous and Mental Disorders

MAIN BUILDING—One of 10 Units in "Cottage Plan"

ST. CROIXDALE ON LAKE ST. CROIX

PRESCOTT, WISCONSIN

Located on beautiful Lake St. Croix, 18 miles from the Twin Cities, it has the advantages of both City and Country. Every facility for treatment provided, including recreational activities and occupational-therapy under trained personnel. Close personal supervision given patients, and modern methods of therapy employed. Inspection and cooperation by reputable physicians invited. Rates very reasonable. Illustrated folder on request.

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Consulting Neuro-Psychiatrists
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there is one tranquilizer clearly indicated **in peptic ulcer...**

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not
increase...

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(BRAND OF HYDROXYZINE)

actually
lowers*
gastric
secretion

*Tests in a series of 25 patients show that there is "a definite and distinct lowering [of both volume of secretions and of free hydrochloric acid] in the majority of patients. . . . No patients had shown any increase in gastric secretions following administration of the drug."[1]

Now you have 4 advantages when you calm ulcer patients with ATARAX:

1. ATARAX suppresses gastric secretions; others commonly increase acidity.
2. ATARAX is "the safest of the mild tranquilizers."[2] (No parkinsonian effect or blood dyscrasias ever reported.)
3. It is effective in 9 of every 10 tense and anxious patients.
4. Five dosage forms give you maximum flexibility.

supplied: 10, 25 and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

references: 1. Strub, I. H.: Personal communication. 2. Ayd, F. J., Jr.: presented at Ohio Assembly of General Practice, 7th Annual Scientific Assembly, Columbus, September 18-19, 1957.

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Secretary

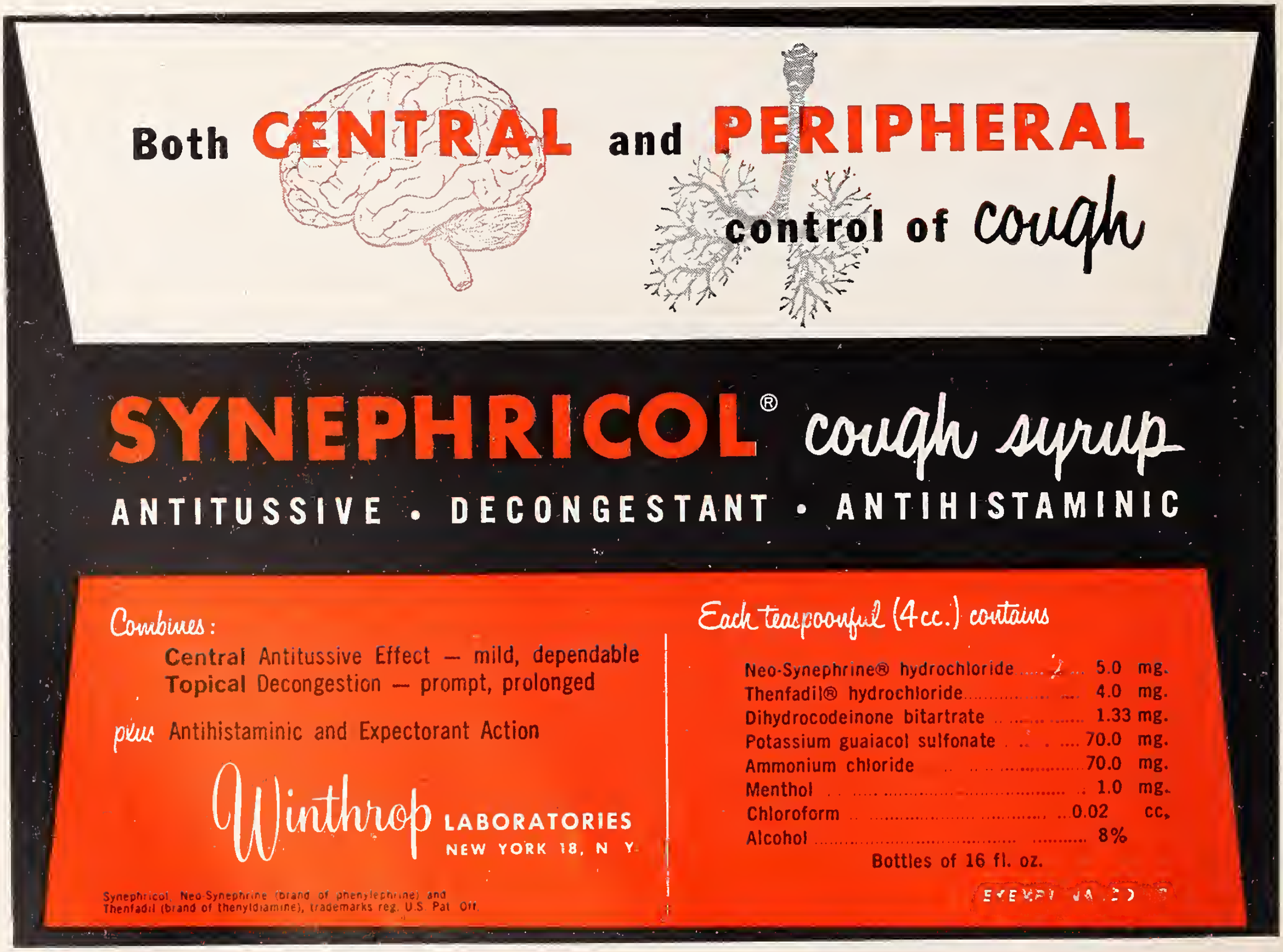
FORMER PHILDELPHIA MAN SENTENCED TO SERVE FIVE-YEAR TERM FOR OBTAINING NARCOTICS BY FRAUD

On January 6, 1958, Simon Manuel Goldberg, thirty-six, 230 Oak Grove Street, Minneapolis, was sentenced by the Hon. Rolf Fosseen, Judge of the District Court of Hennepin County, to serve a term of not to exceed five years in the State Prison at Stillwater for the crime of obtaining narcotic drugs by fraud, deceit, misrepresentation and subterfuge. The defendant, who claimed to have come to Minneapolis recently from Philadelphia, Pa., had obtained eighty-seven tablets of demerol from four Minneapolis physicians by representing that his name was Paul Krueger and that he was suffering from cancer of the stomach and right hip. Goldberg had several scars on his body which he stated were caused by operations for this condition. The defendant had also said that he was a consulting physicist from New York City and that while he was in the Twin Cities in connection with his work he had discovered that he had forgotten to bring with him a sufficient supply of the demerol tablets that his doctor in New York City had been prescribing for him. However, this statement proved to be false.

Two of the Minneapolis physicians who had prescribed demerol for Goldberg became suspicious and notified the authorities after the defendant had left their office but Goldberg was not apprehended until he registered on November 8, 1957, at a Saint Paul hotel, giving his name and address as S. Finley, 240 Central Park South, New York, N. Y. Goldberg was arrested there after he had attempted to obtain narcotic drugs from a Saint Paul physician whom he called to the hotel. Goldberg was then placed in Ancker Hospital, Saint Paul, for observation, and on November 13, 1957, a representative of the Minnesota State Board of Medical Examiners signed a criminal complaint against the defendant which was issued by the Hennepin County Attorney's Office. The defendant waived a preliminary hearing in Minneapolis Municipal Court and then on November 21, 1957, he entered a plea of not guilty before Judge Fosseen in Hennepin County District Court, but on the following day, Goldberg changed his plea to guilty.

The defendant's police record indicates that he first came to the attention of the authorities in July, 1943, when he was held in New York City for a violation of the Selective Service Act. In March, 1949, Goldberg was arrested in Birmingham, Alabama, on a forgery charge and the records show that he was sentenced in Federal Court to serve eighteen months in prison at Tallahassee, Florida. At this time Goldberg was under investigation for impersonating a physicist of the United States Atomic Energy Commission, impersonation of a United States Government employee

(Continued on Page A-68)



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SYNEPHRICOL® cough syrup

ANTITUSSIVE • DECONGESTANT • ANTIHISTAMINIC

Combines:
Central Antitussive Effect — mild, dependable
Topical Decongestion — prompt, prolonged
plus Antihistaminic and Expectorant Action

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Synephricol, Neo-Synephrine (brand of phenylephrine) and Thenfadil (brand of thenyldiamine), trademarks reg. U.S. Pat. Off.

Each teaspoonful (4 cc.) contains

Neo-Synephrine® hydrochloride	5.0	mg.
Thenfadil® hydrochloride	4.0	mg.
Dihydrocodeinone bitartrate	1.33	mg.
Potassium guaiacol sulfonate	70.0	mg.
Ammonium chloride	70.0	mg.
Menthol	1.0	mg.
Chloroform	0.02	cc.
Alcohol	8%	

Bottles of 16 fl. oz.

EXEMPT [illegible]

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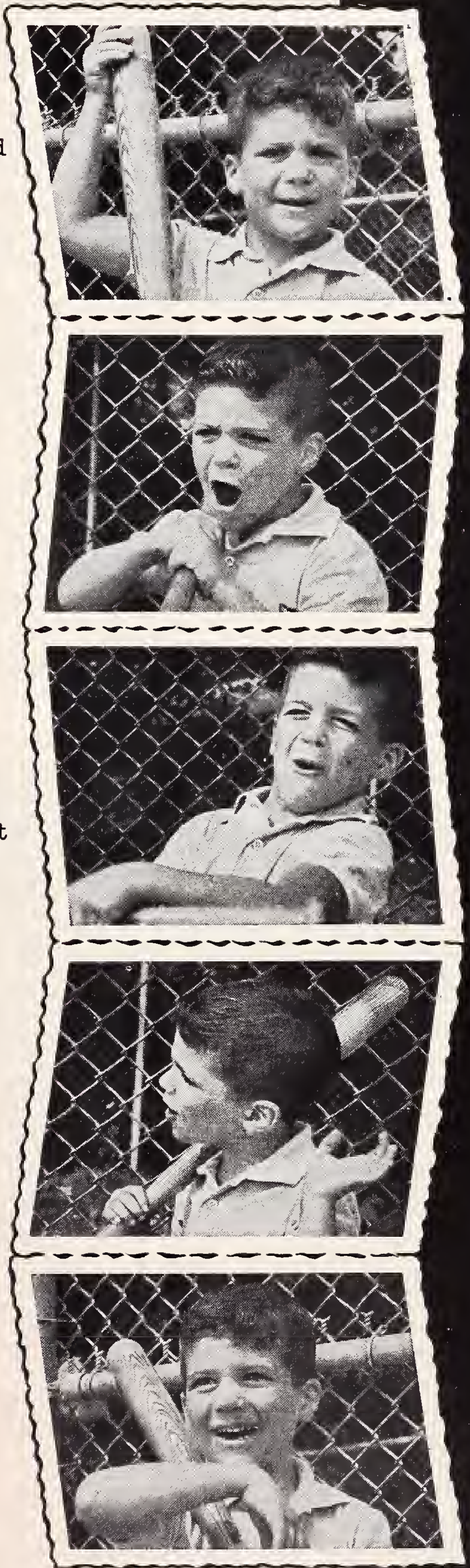

"It happened at work while he was putting oil in something"

"He told Mom his shoulder felt like it was on fire"

"He couldn't swing a bat without hurting"

"But Doctor gave him some nice pills--and the pain went away fast"

"Dad said we'd play ball again tomorrow when he comes home"

AND THE PAIN WENT AWAY FAST

FOR PAIN

Percodan®

(Salts of Dihydrohydroxycodeinone and Homatropine, plus APC)

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ACTS FASTER...
usually within 5-15 minutes

LASTS LONGER...
usually for 6 hours or more

MORE THOROUGH RELIEF...
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New "demi" strength permits dosage flexibility to meet each patient's specific needs. PERCODAN-DEMI provides the PERCODAN formula with one-half the amount of salts of dihydrohydroxycodeinone and homatropine.

AVERAGE ADULT DOSE: 1 tablet every 6 hours. May be habit-forming. Available through all pharmacies.

Each PERCODAN* Tablet contains 4.50 mg. dihydrohydroxycodeinone hydrochloride, 0.38 mg. dihydrohydroxycodeinone terephthalate, 0.38 mg. homatropine terephthalate, 224 mg. acetylsalicylic acid, 160 mg. phenacetin, and 32 mg. caffeine.

Literature? Write

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*U.S. Pat. 2,628,185

(Continued from Page A-66)

with intent to defraud, and falsely obtaining money by impersonating a U. S. Government employee. On January 21, 1950, Goldberg was released on parole from the Federal penitentiary at Lewisburg, Pennsylvania, to which he had been transferred from the prison at Tallahassee, Florida.

In February, 1952, Goldberg was again in the custody of the U. S. Marshall in Washington, D. C., on a check forgery charge and he was sentenced on July 29, 1952, in Federal Court to a prison term of from one to three years. The defendant was sent to the Federal penitentiary at Lewisburg, Pennsylvania, but he was later transferred to the Medical Center for Federal Prisoners at Springfield, Missouri. Subsequently, Goldberg was placed on parole in July, 1953. Goldberg's record reveals that he was again arrested in February, 1955, by the U. S. Marshal in Chicago, Illinois, as a parole violator and returned to the Medical Center for Federal Prisoners from which he was later transferred to the Federal Correctional Institution at Milan, Michigan, where he served the balance of his sentence.

MINNEAPOLIS MAN SENTENCED FOR ILLEGAL SALE OF MEDICINE

On January 3, 1958, Peter J. Toews, seventy-one, 620 South Seventh Street, Minneapolis, was sentenced by the Hon. Victor F. Hanson, Justice of the Peace, in Robbinsdale, Minnesota, for selling a drug and a medicine in a place other than a pharmacy, to a term of thirty days in the workhouse. However, the execution of the sentence was stayed by the Court for a period of one year on condition that the defendant ceases the sale of his products. Toews had been charged with the sale of an alleged vitamin food supplement to a Minneapolis woman for the improvement of her general state of health. The defendant admitted that he had sold two ten-ounce bottles of "Green Life" to the woman for the sum of $8.65.

The activities of the defendant were brought to the attention of the Minnesota State Board of Medical Examiners by a doctor of medicine who learned that the defendant was offering to treat a thirty-year-old Minneapolis man who was suffering from multiple sclerosis. When the matter was investigated it was discovered that Toews had assured the patient that one of his products, "Springreen," would be good for the patient's condition. Toews offered to sell a two-months' supply of the product for $58.00, but the patient declined to make the purchase, saying that he would first like to consult with the physician in charge of his case. When further investigation disclosed that Toews had previously made an actual sale of "Green Life" to the woman referred to above, he was charged with an offense in that case.

The defendant admitted in a signed statement that he was engaged in selling two products, "Green Life," which he sold primarily to chiropractors and osteopaths, and "Springreen," which he sold to the general public. Toews stated that both products were very similar and were vitamin food-supplements. Although Toews claimed to be a retired milling company worker, he indicated that he has been engaged in selling various vitamin products since 1951. Toews further stated that his products were purchased from Springreen Products, Inc., 470 Commonwealth Avenue, Boston, Massachusetts.

TAKE A LOOK AT
NEW DIMETANE
THE UNEXCELLED
ANTIHISTAMINE

SAINT PAUL MAN SENTENCED FOR FORGING NARCOTIC PRESCRIPTIONS

On January 16, 1958 Myron A. Seefeldt, forty-six, 1252 Rice Street, Saint Paul was sentenced by the Hon. Arthur A. Stewart, Judge of the District Court of Ramsey County, to a term of not to exceed five years in the State Reformatory for Men at St. Cloud, pursuant to the defendant's plea of guilty on December 11, 1957, to an information charging him with uttering a false and forged prescription calling for 30 cubic centimeters of demerol. However, Judge Stewart then stayed the execution of the sentence and placed Seefeldt on probation for a period of five years and specified that one of the conditions of probation was to be that the defendant shall refrain from the personal use of narcotic drugs unless such drugs should be prescribed for him by a physician.

Seefeldt was arrested on December 3, 1957, in a Saint Paul drug store after he had presented two forged prescriptions for compounding and had received from the pharmacist the parepectolin and a 30 cc. vial of demerol called for by the prescriptions. When the defendant was searched, one hypodermic syringe, two hypodermic needles and a vial containing approximately 12 cc. of demerol were found. A written statement was taken from the defendant after his arrest and he admitted that he had forged the name of a Saint Paul physician, from whom he had stolen some of the prescription blanks, to numerous prescriptions for parepectolin and demerol. According to Seefeldt, he began using narcotic drugs in 1939. When the records of four Saint Paul pharmacies were inspected, it was discovered that the defendant between August 5, 1957, and December 3, 1957, had forged thirteen prescriptions for parepectolin and fifty-five prescriptions for demerol.

On February 19, 1951, Seefeldt was convicted of the crime of grand theft in the Superior Court of Shasta County, California, for which he was sentenced to serve a three year term in San Quentin Prison. He entered the prison on February 22, 1951, was paroled on May 22, 1952, and was declared a parole violator on July 28, 1953. Upon being questioned by Judge Stewart prior to being sentenced, Seefeldt stated that he was presently working as a machinist in Grantsburg, Wisconsin. The Court was informed that the defendant, who had previously submitted to several operations, was suffering from an ailment for which he was under the care of a physician. Judge Stewart stated that in view of the circumstances of this case he had decided to place the defendant on probation.

Sigmoidoscopy is one of the most important parts of the cancer detection examination, and is too often omitted by the general practitioner.

* * *

The place for the examination of persons with symptoms of cancer is the private physician's office rather than a detection center.

* * *

The few cancers found among asymptomatic persons, as well as the high cost of each examination, preclude large-scale financing of cancer detection centers by either governmental or voluntary agencies.

* * *

A well-trained practicing physician can perform in his office all the procedures that are utilized in the cancer detection center provided he has the necessary equipment which he can readily obtain.

Fundamentally most of the cerebellar tumors in childhood are either astrocytomas or medulloblastomas.

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TABLETS (4 MG.), ELIXIR (2 MG. PER 5 CC.) AND EXTENTABS® (12 MG.) UNEXCELLED POTENCY, UNSURPASSED THERAPEUTIC INDEX AND RELATIVE SAFETY. MINIMUM DROWSINESS AND OTHER SIDE EFFECTS. A. H. ROBINS CO., INC., RICHMOND, VIRGINIA. ETHICAL PHARMACEUTICALS OF MERIT SINCE 1878
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- *Complete Nursing, Medical, Physiotherapy and Occupational Therapy facilities.*
- *Hospitalization, when necessary, is available at Glencoe Municipal Hospital, directly across the street from Glenhaven.*
- *No limitation on age or sex of residents.*
- *Rates and illustrated brochure available on request.*

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Clark W. Truesdale, M. D., Medical Director

General Interest

Lamberton's new resident physician is **Dr. John A. Sturgis.** He will replace **Dr. Morton Roan** who recently retired.

Dr. and Mrs. Roan have taken up temporary residence in Minneapolis where they will remain for several weeks. Although there are no definite plans for the future, the Roans may move to New Mexico where Dr. Roan will continue to practice if his health permits.

* * *

Dr. Hilbert Mark, Minnesota health official, has been named Toledo, Ohio, city health officer.

He has resigned as director of local health administration, the state health department announced.

His successor in the state health department will be **Dr. William C. Harrison,** who has been chief of the section of local health services.

* * *

Dr. Albert J. Schroeder, Minneapolis pediatrician, has been elected chairman of a new medical advisory committee to the Minneapolis public schools health department.

Other committee members, appointed by the Hennepin County Medical Society, are **Drs. Charles Hymes, Harold S. Ulvestad, Frederick G. Rosendahl, Paul W. Linner, William D. Remole, John H. Rosenow** and **James T. Garvey.**

* * *

Dr. Charles Koenigsberger, who has been a physician in Mankato for more than thirty years, was honored by Mankato Clinic doctors and their wives at a party held recently at the Century Club in North Mankato.

* * *

Renville-Redwood Medical Society, at a meeting held recently at Olivia, resolved to resume efforts to get accredited ratings for hospitals at Redwood Falls, Olivia and Sleepy Eye.

The society first voted for the move in May, but plans were deferred here, it was reported, because of labor difficulties. Redwood Falls hospital board in December decided, after a report by **Dr. James B. Flinn,** hospital chief of staff, that accreditation plans not be initiated "at this time."

* * *

Newly elected officers of the Waseca County Medical Society are **Dr. George Olds,** president; **Dr. S. C. G. Oeljen,** vice president; and **Dr. M. C. Florine,** secretary-

(Continued on Page A-73)

when anxiety and tension "erupts" in the G. I. tract...

IN GASTRIC ULCER

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Meprobamate with PATHILON® Lederle

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Dosage: 1 tablet t.i.d. at mealtime. 2 tablets at bedtime. *Supplied:* Bottles of 100, 1,000.

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when you encounter

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- gastrointestinal infections
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infections,
prescribe superior

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Squibb Tetracycline Phosphate Complex

In your patients, **SUMYCIN** *produces:*

1. Superior initial tetracycline blood levels—faster and higher than ever before—assuring fast transport of adequate tetracycline to the site of the infection.

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Supply:	*Tetracycline phosphate complex equiv. to tetracycline HCl (mg.)*	*Packaging:*
Sumycin Capsules (per Capsule)	250	Bottles of 16 and 100
Sumycin Suspension (per 5 cc.)	125	2 oz. bottles
Sumycin Pediatric Drops (per cc.—20 drops)	100	10 cc. dropper bottles

'SUMYCIN' IS A SQUIBB TRADEMARK

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Squibb Quality—
the Priceless Ingredient

(Continued from Page A-71)

treasurer. The wives of each officer will hold the corresponding position in the association's auxiliary.

* * *

St. Luke's Hospital new chief of staff is **Dr. Joseph F. Bicek.**

Dr. Charles T. Enginton is chief of staff-elect and **Dr. Milton Sorem** is secretary of the medical staff. They were elected at the annual meeting of the hospital's medical staff. Department heads elected for 1958 are: **Dr. James A. Bellomo,** chief of medicine; **Dr. Charles J. Mishek,** chief of general practice; **Dr. Darrell E. Westover,** chief of surgery, and **Dr. Thomas K. Krezowski,** chief of obstetrics-gynecology.

* * *

Minneapolis Swedish Hospital's newly elected Chief of Staff is **Dr. J. Harold Schultz.**

Dr. Wayne C. Rydburg was elected vice president and **Dr. William H. Anderson** was re-elected secretary-treasurer.

* * *

Dr. John Dwyer has been elected chief of staff at Duluth's Miller Memorial Hospital. He succeeds **Dr. Kenneth Teich.**

Other new officers are **Dr. Kenneth Storsteen,** vice chief of staff; **Dr. Harold Walder,** staff secretary. **Dr. Henry Bakkila** and **Dr. Teich,** members of the executive committee. **Dr. Mario M. Fischer,** Duluth and St. Louis county health director, is an ex-officio member of the executive committee.

* * *

"Why the Multiple Health Drives?" was the topic of an address presented by **Dr. John F. Briggs** to the Child Psychology Study circle at its meeting in the Groveland Park School in St. Paul.

* * *

Mayo Clinic doctors have been busy attending national medical and allied field meetings during the past month.

Dr. J. A. Ulrich attended a meeting of the Microbiology Study Section of the National Institutes of Health in New Orleans, January 9-11.

Papers were presented at a meeting of the Neurosurgical Society of America, held at Key Biscayne, Florida, January 15-18, by **Drs. C. B. Holman, C. S. MacCarty** and **Alfred Uihlein. Dr. H. J. Svien** presented a movie which he produced in collaboration with **Dr. Matthew W. Wood. Dr. H. R. Terry** also attended.

Dr. MacCarty's paper was written with **Drs. Milam Leavens, J. G. Love** and **J. W. Kernohan,** and Dr. Uihlein's paper was written with **Drs. H. L. Parker, Alexander Schirger** and **J. W. Kernohan.**

* * *

The Olmsted County Tuberculosis and Health Association became incorporated and adopted by-laws at a meeting of the officers and directors held recently at the Public Health Center.

Under the new incorporation, anyone in the county

when anxiety and tension "erupts" in the G. I. tract...

IN DUODENAL ULCER

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may apply for membership in the organization and vote at the annual meeting. Heretofore the association has functioned entirely under contract of the State TB Association.

More active interest and participation by residents of the county is desirable, the board stated. The affairs of the county association have gone well, showing a healthy growth of funds available and effective expansion of activities, but more widespread personal interest is sought, however.

The articles provide for rotation of officers and directors and the election of three directors from the county outside of Rochester. Limits were also placed on terms of service.

The previous officers were all re-elected for one-year terms and board of directors was chosen for the 1958-59 year.

Dr. H. Z. Giffin was re-elected president, and other officers renamed are **Dr. C. H. Hodgson**, first vice president; Forest J. Bandel, second vice president; Mrs. E. J. Pomerleau, secretary, and H. E. Tubbs, treasurer. All are of Rochester.

Elected to one-year terms on the board were **Dr. Charles W. Mayo**, **Dr. John S. Lundy** and Mrs. C. F. Stroebel of Rochester and Mrs. Floyd Stocker of Eyota; two-year terms, Mrs. George B. Logan, **Dr. Viktor O. Wilson** and **Dr. John E. Verby** of Rochester and Mrs. Stanley Hall of Stewartville; three-year terms, **Dr. H. W. Schmidt**, **Dr. David T. Carr** and **Dr. Alfred G. Karlson** of Rochester and **Dr. A. F. Risser** of Stewartville.

Headquarters for the TB and Health Association will now be at the Public Health Center on Fourth Street S.E. in Rochester.

* * *

Dr. W. A. Fansler of Minneapolis, who is on a world tour, gave a talk Friday, February 21, at the University of Witwatersrand, Johannesburg, South Africa, on the topic, "Current Investigations of Polyps of the Rectum and Colon." He presented the same subject on Monday, February 24, at a meeting of the Society of Consultants in Bulawayo, Southern Rhodesia.

Dr. and Mrs. Gordon R. Kamman of St. Paul have recently returned from a six weeks' cruise aboard the S.S. President Wilson. The cruise included stop-overs in China, Japan and Hawaii.

* * *

Drs. R. D. Hanover and **C. B. Will**, International Falls physicians, has moved to quarters in the new building which they constructed at 407 Fourth Street.

The one-story building 37x80 feet, has a decorative brick front set off with large double windows. The structure is built of concrete blocks.

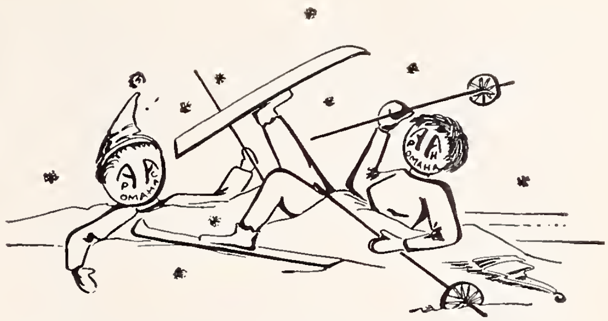
* * *

Dr. Jacob Biedermann, Thief River Falls physician for more than forty years, has discontinued practice due to failing health.

* * *

Dr. John K. Butler, Cloquet, will be installed as chief of medical staff at St. Mary's hospital in Duluth. He succeeds **Dr. D. W. Wheeler**, Duluth.

Dr. Butler, a long-time resident of Duluth, is a graduate of the University of Minnesota medical school and served his internship in St. Mary's Hospital. He has been secretary of the medical staff and was the first member of the staff to serve as chief of the general practice division when it was started in 1953. He is a member of the executive committee of the St. Louis County Medical Society and past president of the Du-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luth chapter of the American Academy of General Practice. He represents the Minnesota State Medical Association on the World Health Committee of the American Medical Association.

* * *

Dr. Russell T. Brown has purchased the Browerville Clinic from **Dr. A. J. Lenarz.**

* * *

Dr. J. Arnold Bargen, chairman of four sections of medicine in the Mayo Clinic, Rochester, and professor of medicine in the Mayo Foundation, Graduate School, University of Minnesota, is vice chairman of the World Congress of Gastroenterology to be held in Washington, D. C., May 25 to 31, 1958.

MEDICAL BUILDING
LUTHER SCHEFFY

"FOURTH FLOOR!... NEURASTHENIA, PSYCHASTHENIA, SCHIZOPHRENIA . ."

* * *

Dr. Bert Eric Schwarz, a fellow in psychiatry of the Mayo Foundation at Rochester from 1951 to 1955, and **Dr. Bartholomew A. Ruggieri,** a fellow in pediatrics at Mayo Foundation from 1952 to 1955, are the authors of a new book for the public entitled "Parent-Child Tensions," published in January, 1958, by the J. B. Lippincott Company of Philadelphia. The book is based on the principles of collaborative psychiatric therapy evolved by Dr. Adelaide M. Johnson, of Rochester, clinical professor of psychiatry in the University of Minnesota. Dr. Schwarz and Dr. Ruggieri are engaged in private practice in Montclair, New Jersey.

* * *

Dr. Harold F. Wahlquist, Minneapolis, was recently named to a new community health board to organize a city-county psychiatric clinic at Minneapolis General Hospital.

* * *

Dr. James Rogers Fox, Minneapolis, was a guest speaker at the meeting of the Zumbrota PTA, early in February. His subject was "Public Health Matters Parents Should Know."

* * *

The following are the officers of the Medical Staff of the St. Cloud Hospital for the year 1958: Dr. L. A. Veranth, Chief of Staff; Dr. C. J. Luckemeyer, Vice President; Dr. R. J. Cesnik, Secretary, and Dr. J. P. O'Keefe, President-Elect.

* * *

Dr. William Sawyer Eisenstadt attended the meeting of the American Academy of Allergy held in Philadelphia, February 1 through 5. During the past year, he served on both the Committee on Aero-Allergens and the Sub-Committee on Weather of the Research Council of the American Academy of Allergy.

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Book Reviews

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COMMON CONTAGIOUS DISEASES. By Philip Moen Stimson, A.B., M.D., Professor of Clinical Pediatrics, Cornell University Medical College, Consulting Pediatrician, New York and Roosevelt Hospitals, Hospital for Special Surgery, et cetera; and Horace Louis Hodes, A.B., M.D., Pediatrician-in-Chief and Director, Department of Pediatrics, Mt. Sinai Hospital, New York; Clinical Professor of Pediatrics, College of Physicians and Surgeons, Columbia University, et cetera. 5th edition. 624 pages. Illus. Price, $8.50. Philadelphia: Lea & Febiger, 1956.

This is the fifth edition of this well-written text or manual first published in 1931. The book has been very thoroughly rewritten for this last edition to bring it up-to-date in every way. It is a very readable and handy text on all aspects of common contagious diseases. The style of the book, a carefully organized table of contents, a cleverly written text, a glossary, index, and even a terminal handy summary chart in the inside of the back cover make it a splendid book for any general practitioner or pediatrician to have in his office. The illustrations and plates are very well reproduced.

R. O. Bergan, M.D.

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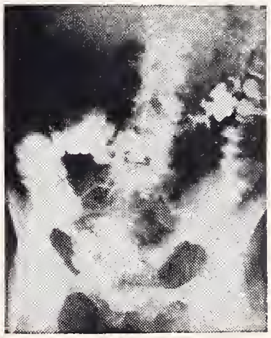

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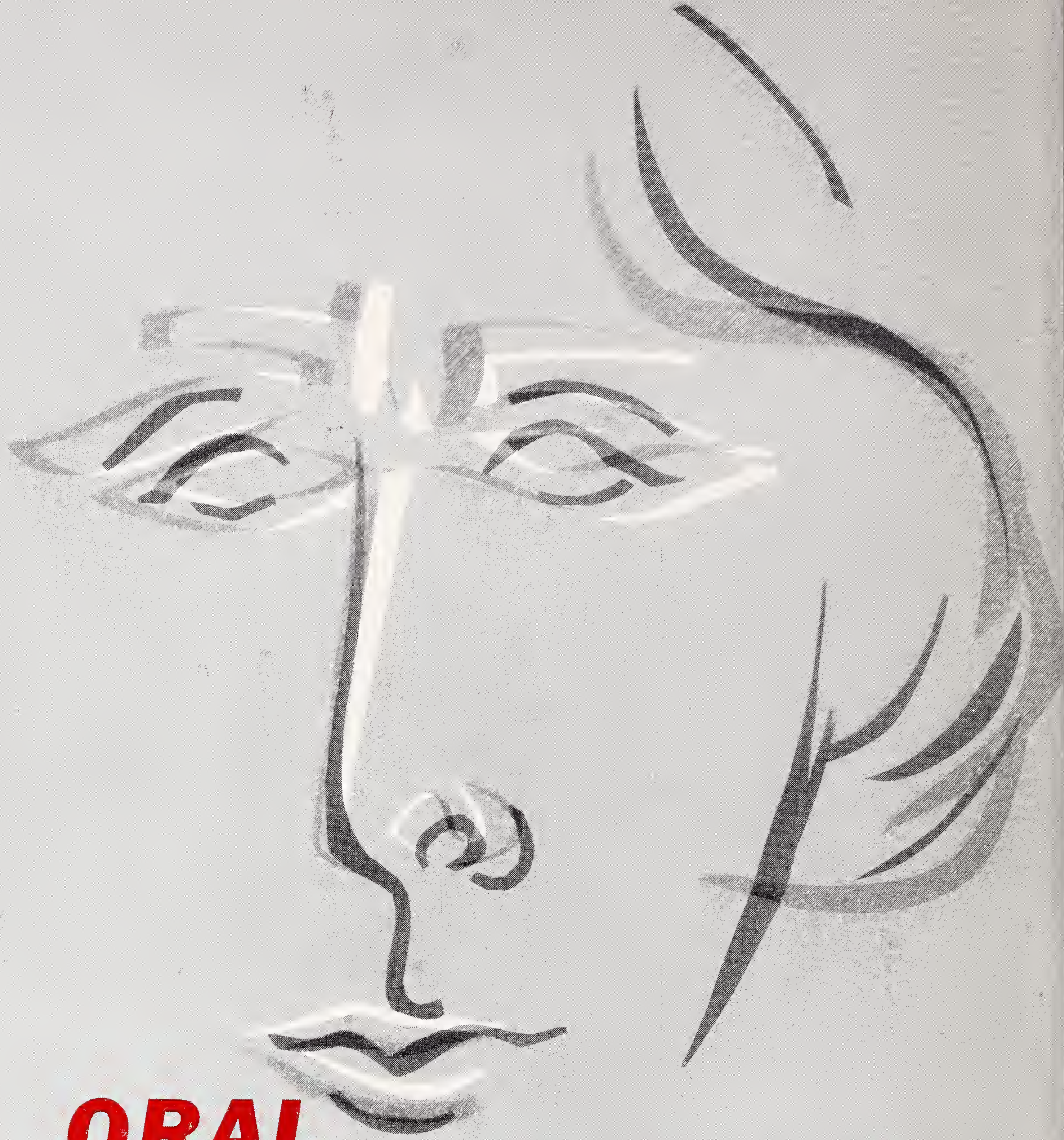

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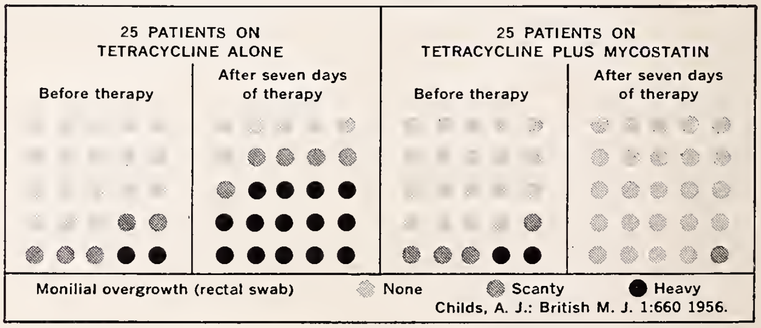

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Original Contributions

Value of Central and Peripheral Intra-arterial Pressures and Pulse Contours in Cardiovascular Diagnosis

J. LEO WRIGHT, M.D.
EARL H. WOOD, M.D., Ph.D.
Rochester, Minnesota

INTRA-ARTERIAL pressure was first measured in man in 1856 by Jean Faivre,[1] a senior intern at the Hôtel Dieu in Lyon, France. The pressure in the arteries of three patients undergoing amputation of limbs was measured by connecting the proximal end of the cut artery to a U-shaped mercury manometer similar to the type previously used by Poiseiulle. The subsequent history of the measurement of intra-arterial pressure and pulse contours for diagnosis is really a history of the introduction and improvement of instruments and recording methods designed for the faithful reproduction of pressure and pulse contours.[2-7]

Until the 1930's, however, the clinical use of intra-arterial pressure measurement in man was infrequent and was conducted with caution during blood transfusions, during amputations[8] and in terminally ill patients.[9] In 1931, Wolf and von Bonsdorff[10] reported study of pressures recorded by means of percutaneous needle puncture of the brachial artery in fifteen persons; in 1932, Dameshek and Loman[11] reported similar measurements in approximately 150 patients in a state hospital. The primary purpose of these studies was a comparison of intra-arterial pressures with the readings obtained by indirect cuff methods.

Since 1946, more than 2,000 percutaneous punctures of the radial artery[12] have been done at the Mayo Clinic in the Cardiovascular Laboratory, Section of Physiology. This work includes normal persons, infants and adults undergoing diagnostic cardiac catheterization, as well as patients undergoing special studies of intra-arterial pressure. The sequelae of arterial puncture have been minor. Careful periarterial infiltration with procaine has controlled pain adequately. Hematoma, a potential complication, has not been encountered when pressure over the artery is maintained after withdrawal of the needle. Minimal tenderness over the site of puncture has been experienced by some patients for twenty-four to forty-eight hours.

During cardiac catheterization, indwelling intra-arterial needles are routinely used for monitoring pressure and for withdrawing samples of blood for oxygen determination at rest and during exercise.[13] Intra-arterial needles are also used for continuous recording of arterial indicator-dilution curves[14,15] and may be left in place for hours without discomfort or hazard to the patient. Obvious additional clinical applications are determinations of intra-arterial blood pressure in extremely obese patients or patients who have severe arteriosclerosis of peripheral arteries, both of which groups tend to give unreliable readings of blood pressure when it is determined by the indirect method. Other particularly valuable uses of this technique are (1) continuous monitoring of blood pressure during operations on the heart,[16,17] (2) measurement of the pressure distal to the aortic valve during catheterization of the left side of the heart[18] and (3) localization of abnormal obstructions of the circulation, which will be discussed later in the text.

Methods

Pressure pulses were recorded utilizing photokymographic recording methods with strain-gauge manometers.[7] In peripheral arteries, such as the radial, brachial, femoral, dorsalis pedis or posterior tibial, pressures were obtained by direct arterial puncture using special, internally polished,

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Presented at the Symposium on Peripheral Vascular Disease co-sponsored by the Minnesota Heart Association and the Mayo Foundation, Rochester, Minnesota, September 23, 1957.

Dr. Wright, former Assistant to the Staff, is now Director, Cardio-Pulmonary Laboratory, Baptist Memorial Hospital, Memphis, Tennessee. Dr. Wood is from the Section of Physiology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

20-gauge needles. Arterial catheters were used for the simultaneous recording of central pressures in the aorta and branches of the aorta. These catheters, which have internal and external diameters of 0.5 and 0.8 mm., respectively, can be advanced readily into the arterial system through thin-walled 19-gauge needles after puncture of the radial or femoral artery. Larger catheters similarly introduced and routinely used for physiologic studies requiring the injection of dyes are equally suitable for recording arterial pressures.

For practical purposes, optimally damped recording systems having a uniform response to frequencies up to 10 cycles per second (cps) are adequate for recording arterial pressure, although manometer recording systems having response characteristics of ± 10 per cent of the static sensitivity up to 40 cps are routinely used in the laboratory.[19-21]

Pulse Contours in Healthy Persons

The differences in pressures and pulse contours generated by the same heart beat when recorded simultaneously at multiple sites in a normal adult are shown in Figure 1. It will be noted that the contour of the aortic pulse undergoes pronounced transformation as it moves peripherally in the vessels of the arms; it also undergoes changes th are not exactly similar as it moves peripherally i the lower extremities. At the same time, in portant changes in pressure occur. It is evide that the systolic pressure increases as the puls wave moves peripherally. However, the diastoli and mean pressures decrease slightly.

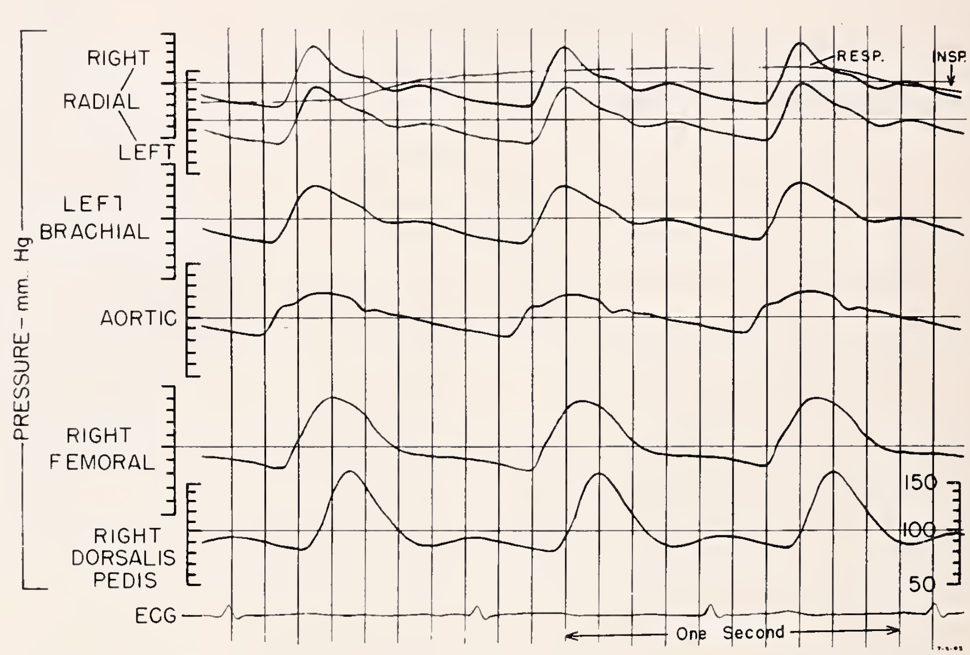

Fig. 1. Pulse contours in a healthy thirty-one-year-old man. Pressure pulses were recorded simultaneously in the right and left radial, left brachial, aortic arch, right femoral and right dorsalis pedis arteries. A reference line (100 mm. of mercury) is drawn through each series of pulses. The contour of the aortic pulse is significantly changed as the pulse wave moves peripherally into the arm. Somewhat different changes are observed as the pressure pulse generated by the same heartbeat moves peripherally into the leg. The associated increase in systolic pressure is greater in the more distally located dorsalis pedis artery than in the radial artery.

Figure 2 is a simplification of the previou figure in that only pressure pulses recorded be tween the aortic arch and the radial artery ar shown. These contours were obtained in a health person by means of an arterial catheter introduce via a needle in the radial artery. Aortic pressur recorded by a second catheter introduced throug a needle in the femoral artery and the radial pressure in the opposite arm were used as control during the gradual withdrawal of the firs catheter. The subclavian contour is characterize by an initial wave, a plateau type of systolic maximum, followed by a dicrotic halt, after which i seen a small postdicrotic wave. As the pulse wav moves out into the arm, the initial wave become steeper and larger in magnitude, the maxima pressure becomes peaked and the dicrotic hal tends to move down and out. At the radial artery the wave that occurs between the systolic maxium and the dicrotic halt appears to be a remant of the plateaulike maximum of the subavian contour. The small aortic postdicrotic ave disappears shortly after the pulse wave ters the subclavian-radial system.

In Figure 3, simultaneously recorded radial and rtic pressure pulses in a healthy person are perimposed to illustrate the changes in pressure d contour between the two sites. Since the two rain gauges used to record the aortic and radial ulses had the same sensitivities, the calibration pplies to both. Note the significantly greater rtic diastolic pressure. In this person, the radial stolic pressure was approximately 12 per cent reater than the aortic systolic pressure. In fteen healthy persons, the radial systolic pressure veraged 12 per cent greater than did the simulneously recorded aortic systolic pressure, the nge being from 2 to 21 per cent. The diastolic d mean pressures decreased an average of 4 per nt, the ranges being –10 to 0 and –9 to –1 per nt, respectively. Similar results from a different ries of persons have been obtained previously this laboratory.[22]

Pulse Contours in Cardiovascular Diseases

Valvular Aortic Stenosis.—In Figure 4, the rtic and radial contours in a patient who had vere valvular aortic stenosis are superimposed. he aortic diastolic pressure is slightly greater than e radial diastolic pressure, as in normal persons. nlike the normal state, however, the two conurs are remarkably similar, an abnormal acrotic pause is seen on the radial contour, the crease to the systolic maximum is greatly pronged in both pressure pulses and, in pronounced ntrast to the normal state, the radial systolic ressure is less than the aortic systolic pressure. inally, a postdicrotic wave is not identifiable on e aortic contour. Our observations indicate that e central postdicrotic wave is present on presre pulses recorded near a normally functioning rtic valve but that it usually is absent in patients ho have significant aortic valvular disease.

Subvalvular Aortic Stenosis.—In Figure 5, presre pulses obtained during catheterization of the ft side of the heart in a patient with valvular rtic stenosis are compared with similar contours om a patient with subvalvular aortic stenosis. vidence of severe obstruction to left ventricular tflow is present in both patients. The radial pulses are equally abnormal in contour. The aortic contour in the patient with subvalvular stenosis resembles a normal contour to a surprising degree. The postdicrotic wave on the aortic contour, which suggests a normally functioning aortic valve in the patient with subvalvular aortic stenosis, is absent in the patient with valvular stenosis. These diagnoses were confirmed surgically, and both lesions were successfully corrected.

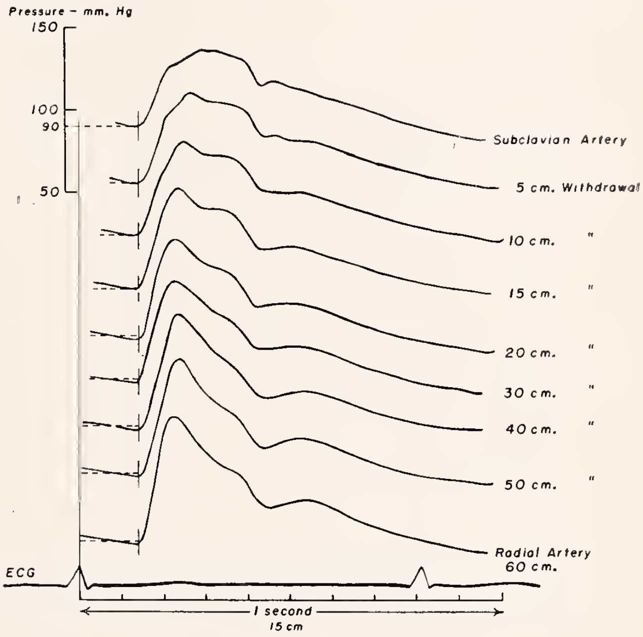

Fig. 2. Pulse contours in a healthy thirty-year-old man, showing transformation of the pressure pulse in the subclavian-radial system. Pressure pulses were recorded consecutively during withdrawal of the tip of an arterial catheter from the subclavian artery near the aorta to the radial artery in the left arm. The onset of each pressure pulse is aligned for purposes of comparison. As the pulse wave moves peripherally, the initial wave steepens and increases in magnitude, the dome-shaped systolic maximum becomes peaked, the dicrotic halt moves down and to the right and becomes slurred. The low-amplitude, central postdicrotic wave is not seen after the catheter has been withdrawn 10 cm. or more. The prominence of the radial dicrotic wave is due, in part, to the change in position of the dicrotic halt (see Figure 3). The horizontal dashed line intersecting the onset of each pulse contour is the calibration reference point (90 mm. of mercury). The interval of time from the peak of the R wave of the electrocardiogram to the onset of the systolic upswing of each pulse wave is indicated by the duration of each tracing to the left side of the short vertical lines, which mark the onset of systole for each pulse. These pulses are photostatic copies of pulses selected from the original photographic records on the basis that the contours of the aortic pressure pulses recorded simultaneously from a second arterial catheter, the phase of the respiratory cycle and the electrocardiogram were all closely similar.

Aortic Insufficiency.—In Figure 6, simultaneously recorded aortic and radial pressure pulses in a patient who had severe aortic insufficiency are superimposed. The aortic diastolic pressure, as in the normal state, is slightly greater than the radial diastolic pressure. The diagnostic features of aortic insufficiency are (1) a low diastoli pressure, in this instance less than 50 mm. of mercury, (2) a wide pulse pressure and (3) a rapid rise to a peaked radial systolic pressure. In addi-

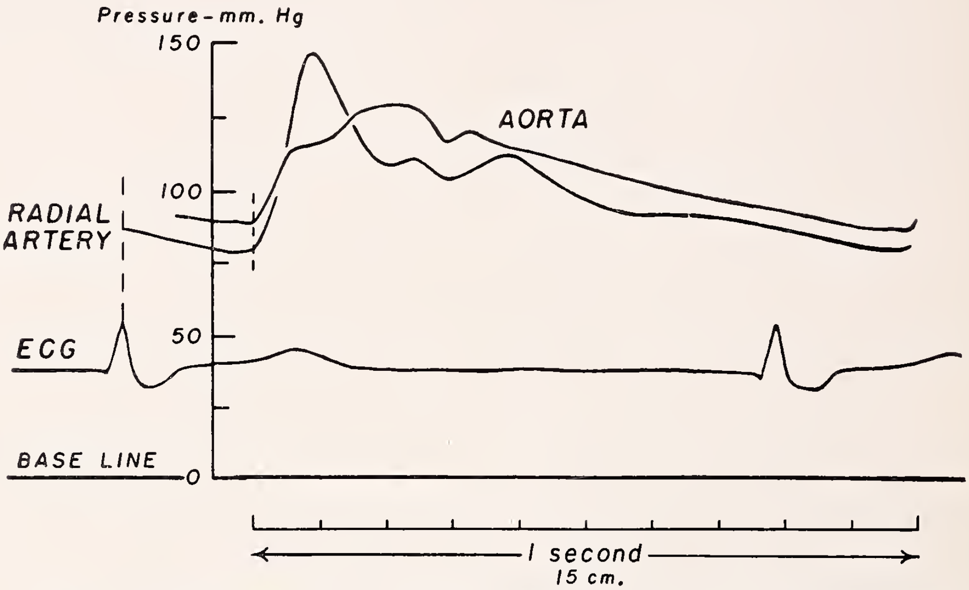

Fig. 3. Pulse contours in a healthy thirty-year-old man. Pressure pulses were recorded simultaneously in the aorta and radial artery, utilizing strain gauges having equal sensitivities. For purposes of comparison, photostatic copies of individual pulses were superimposed, with the onset of the pressure pulses aligned. The time interval between the peak of the R wave of the electrocardiogram and the beginning of the recording of the aortic pulse approximates the transmission time from the aorta to the radial artery. The increase in systolic pressure at the radial artery is the only portion of the pressure wave that exceeds the simultaneously recorded aortic pressure pulse. The remaining elements of the radial pressure wave are less than the aortic pressures. The pressure pulses shown in Figures 2 and 3 were recorded from different persons.

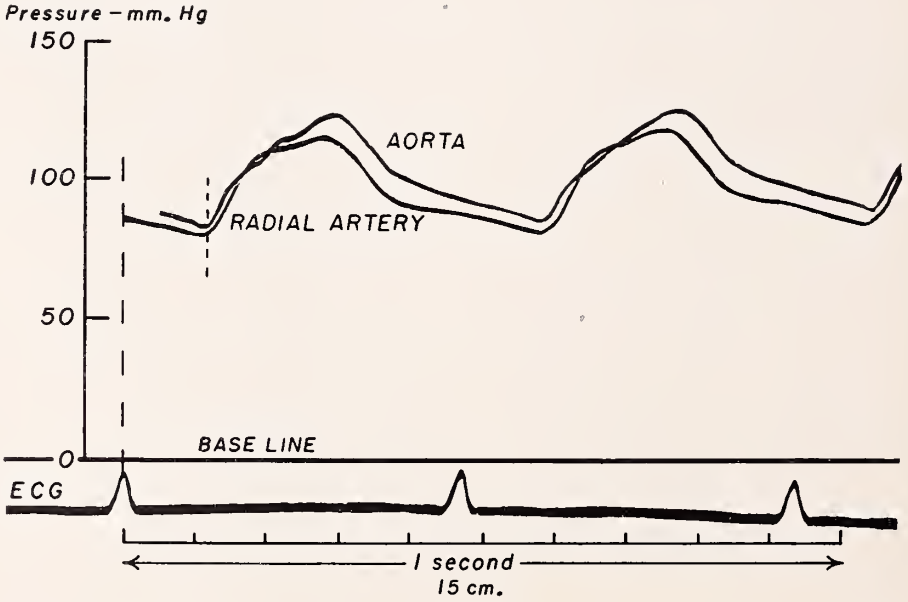

Fig. 4. Pulse contours in a twelve-year-old boy who had severe valvular aortic stenosis. The simultaneously recorded aortic and radial pressure pulses have been photostated and superimposed as in Figure 3. Note the similarity of the two contours, the decreased systolic pressure, the anacrotism of the radial pulse (lower tracing) and the absence of the central postdicrotic wave of the aortic contour (compare with Figures 2 and 3).

tion, the absence of both a well-defined dicrotic halt and a postdicrotic wave on the aortic contour strongly supports the diagnosis of aortic insufficiency.

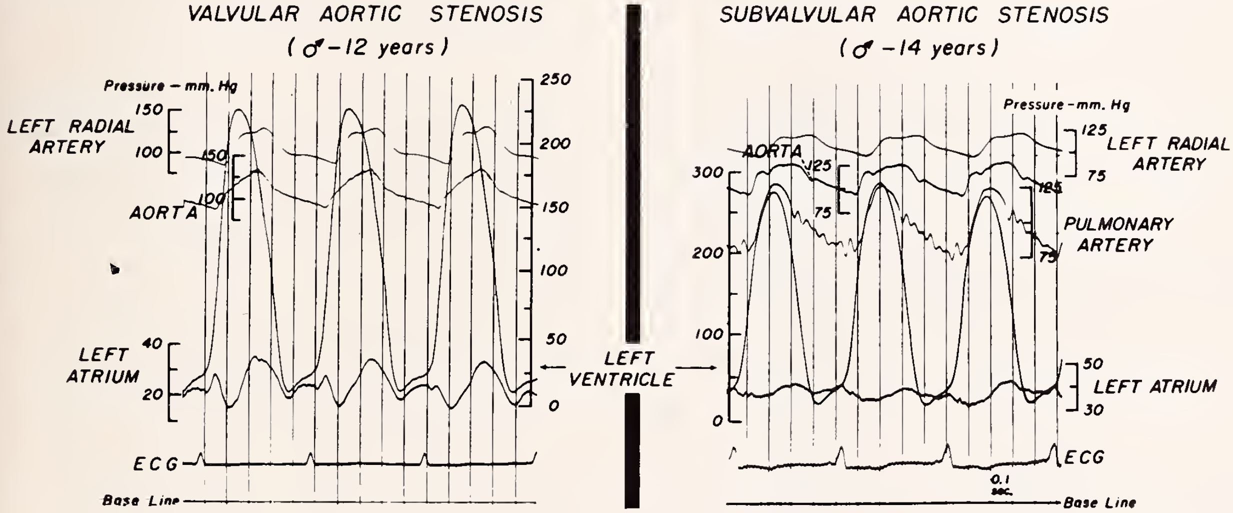

Fig. 5. Pulse contours in a twelve-year-old boy with valvular aortic stenosis compared with similar contours in a fourteen-year-old boy with subvalvular aortic stenosis. Simultaneously measured radial, aortic, left ventricular and left atrial pressures were recorded during percutaneous catheterization of the left side of the heart. The left ventricular systolic pressure is 125 to 150 mm. of mercury greater than the aortic systolic pressure in both patients, indicating severe obstruction to left ventricular outflow. Pronounced anacrotism of both radial pulse contours is evident. However, the aortic pressure pulse in the patient with subvalvular aortic stenosis resembles a normal aortic contour. In addition, a small central postdicrotic wave is easily identified; while not a proved diagnostic sign, this finding suggests the presence of a normally functioning aortic valve. Note the absence of left atrial-left ventricular pressure differences across the mitral valve during diastole, which excludes the presence of mitral stenosis.

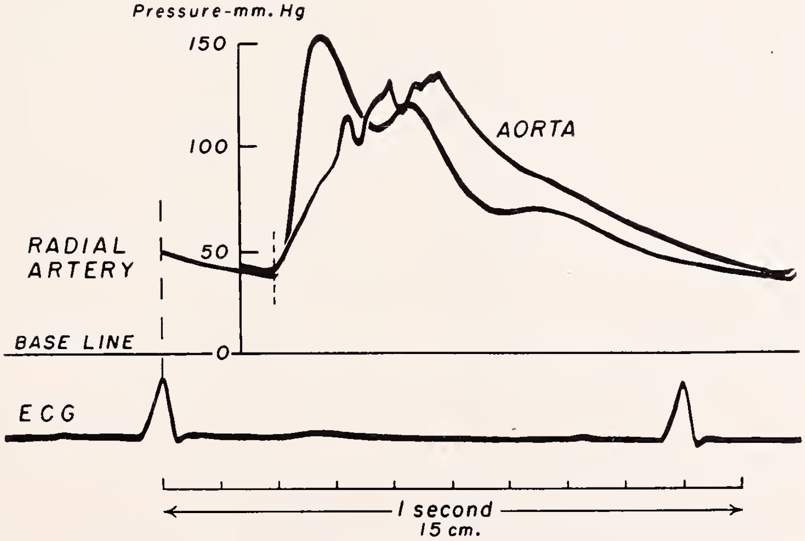

Fig. 6. Pulse contours in a twenty-four-year-old man who had aortic insufficiency. Aortic and radial pressure pulses were recorded simultaneously, utilizing strain gauges having equal sensitivities. The pulses have been photostated and superimposed for comparison as in Figure 3. The contours are characterized by a low diastolic pressure and wide pulse pressure. The radial upstroke is abrupt. The dicrotic halt of the aortic contour is slurred, and a well-defined postdicrotic wave cannot be identified.

Coarctation of the Aorta.—Obstruction in the aorta caused by coarctation produces characteristic changes in pressures and pulse contours. Figure 7 compares the preoperative radial and femoral pressures with similar pressures recorded in the same patient two weeks and again five years after operation for coarctation. The classic findings in coarctation of the aorta are (1) hypertension in the upper extremities, (2) a characteristic "saw-tooth" contour of small amplitude in the tracing from the femoral artery, (3) a delay in arrival of the onset of the pulse at the femoral artery and (4) a great decrease in the ratios of femoral/radial systolic and pulse pressure to far below the range of normal. The significant decrease in radial systolic and diastolic pressures two weeks after operation is followed by a further striking decrease in radial pressure during the next five years. Concomitantly, a change toward normal occurs in the femoral contour and pressures, so that essentially no difference exists between the pressures in radial and femoral arteries at the five-year study. Another record of the changes caused by surgical correction of coarctation is seen in Figure 8. In a group of ten patients studied preoperatively and again four to seven years after resection of the site of coarctation and end-to-end anastomosis of the aorta slight but continued improvement in the pressures measured above and below the site of resection was demonstrated.[23]

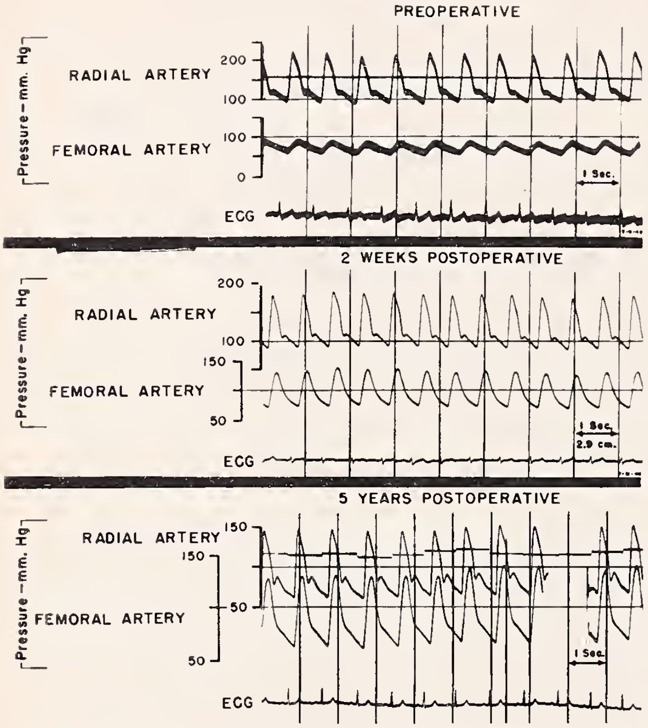

Fig. 7. Pulse contours in coarctation of the aorta. Radial and femoral pressure pulses were recorded simultaneously above and below the site of coarctation at a paper speed approximately one sixth as fast as in the other figures. *Upper panel (preoperative):* hypertension above the coarctation is associated with the characteristic "saw-tooth" femoral contour. The pulse pressure of the latter is small, and the diastolic pressure is less than the diastolic pressure above the coarctation. *Middle panel (two weeks after operation):* after resection of the coarctation and end-to-end anastomosis of the aorta, the hypertension above the site of resection is slightly less. The femoral pulse has changed from a "saw-tooth" appearance toward a more normal contour. However, both the systolic and diastolic pressures are significantly less than the radial pressures. *Lower panel (five years after operation):* the radial and femoral contours are within the range of normal. The systolic and diastolic pressures above and below the resected zone are now essentially the same.

Systemic Hypertension.—In Figure 9, the radial and aortic pressures are compared in a patient who had hypertension caused by unilateral renal disease. The intra-arterial pressures recorded preoperatively ranged from 190/130 to 210/140. Following nephrectomy, this patient became normotensive.

No characteristic diagnostic changes have been recognized in central or radial pulse contours in the limited number of patients with systemic hypertension studied in the laboratory. Several nonspecific abnormalities in pulse contour in Figure 9 are noted. The radial contour is abnormal and, when viewed alone, might suggest valvular aortic stenosis. Viewed together with the normal aortic contour, especially when associated with an absence of amplification of the systolic pressure at the radial artery, it might suggest subvalvular aortic stenosis. Against this are the severe hypertension, wide pulse pressure, rapid build-up time and the large amplitude of the initial upstroke of the radial pulse, features not seen in aortic stenosis.

Comment and Summary

The mechanism controlling transformation of pressure pulse contours during transmission to the peripheral arterial system and the explanation of the amplification of systolic pressure remain obscure. These appear to be related phenomena in the aortic-radial and aortic-femoral systems and are difficult to explain fully by a "standing-wave" hypothesis.[24] It is probable that these phenomena are related to the resonant and damping characteristics of the arterial system.[25,26] In healthy persons, a change of contour between the aorta and the radial artery usually is associated with amplification of systolic pressure at the radial artery. In aortic stenosis and some isolated instances of other conditions (Fig. 9), absence of an increase in systolic pressure at the radial artery is associated with absence of transformation in pulse contour.

In our experience, the reliability of pulse contours for diagnosis has been considerably enhanced by simultaneous recording of central and periphral pressure pulses. Once the mechanism or nechanisms controlling the transformation of ulse contours and the amplification of systolic ressure are known, it is likely that the recording f intra-arterial pressure will become an even harper diagnostic tool.

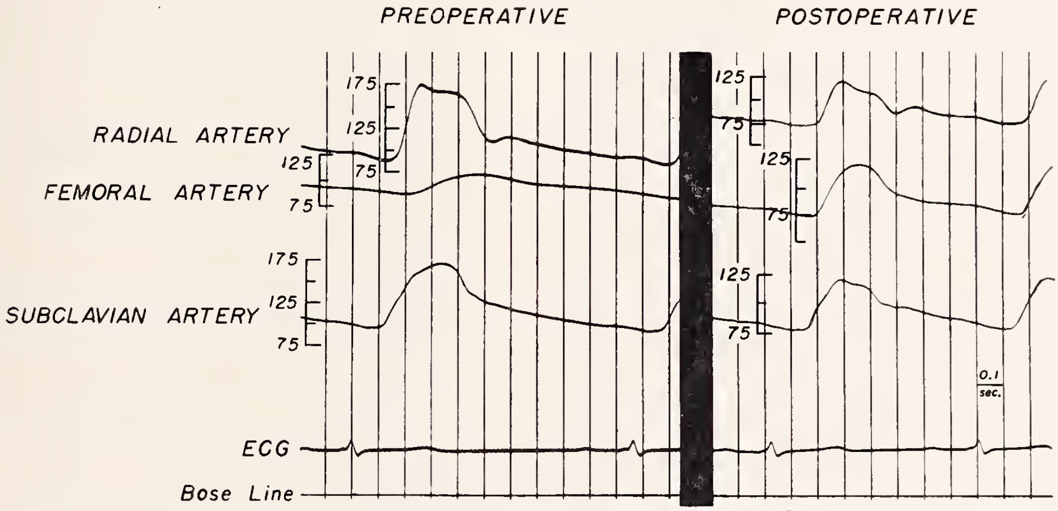

Fig. 8. Comparison of central (subclavian) and peripheral pressure pulses in coarctation of the aorta before and after surgical correction.

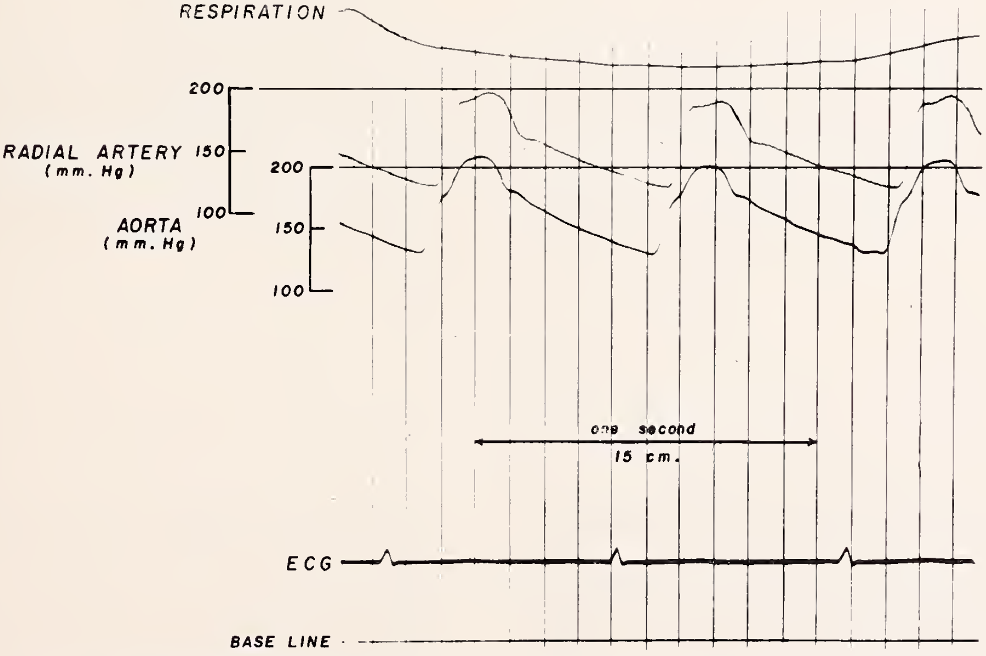

Fig. 9. Simultaneously recorded aortic and radial pulse contours in a nine-year-old boy with systemic hypertension caused by unilateral renal disease. Note the anacrotic radial pulse and absence of an increase in systolic pressure at the radial artery (see text).

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STUDY ON HEART DISEASE

Heart attacks strike far more often when a person is resting than when he is working. Approximately 30 per cent of all coronary heart attacks occur when people are resting; a mere 2 per cent when they are engaged in sports, running, lifting or moving a load. Several studies indicate that the highest death rate in men occurs among those employed at sedentary jobs.

Moreover, very few cardiac patients need to curtail their activities drastically. The majority of persons disabled by heart disease recover from attacks to live many years and most are able to resume work. Less than 1 per cent of all victims of heart disease require complete rest.

Studies show that deaths from heart disease tend to rise during the winter months and to decline during the summer.

They further reveal that heart disease takes a far greater toll among men than women—a difference which has been steadily mounting over the past few decades. Thus, within the last forty years, the heart disease death risk within the forty-five to sixty-five-year age group has increased nearly 25 per cent for men and decreased at almost the same rate for women.

Death rates for heart disease vary, too, from state t state, with the New England states heading the list an southwestern states, such as Arizona and New Mexico ranking last.

As far as classification is concerned, hypertension an arteriosclerosis account for over 90 per cent of death for cardiovascular disease, including vascular lesions af fecting the central nervous system and myocardium Coronary heart disease remains the leading heart problem

However, with adequate care, 22 per cent of patient with heart disease can expect improvement or halting o progress of the disease, compared with only 2 per cen improvement for those without care.

The need for adequate treatment is underscored by th fact that deaths from cardiovascular disease are risin rapidly. The proportion of deaths due to cardiovascula disease has increased from about 20 per cent in 1900 t almost 55 per cent in 1956.

Moreover costs for the disease have been increasin dramatically. They now amount to more than $3 billio worth of working time in the United States, and a loss in income tax to the Federal Government of almos $4 million.—*Patterns of Disease.*

oninflammatory Diseases of Smaller rteries and Arterioles

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ALTHOUGH the smaller blood vessels constitute a large part of the blood vascular stem, there has been little knowledge of or terest in their diseases until relatively recently. ven now the state of the smaller blood vessels such well-recognized conditions as thrombongiitis obliterans and arteriosclerosis obliterans largely unknown. Atheromas have been idenfied in arteries as small as 600 to 1000 microns diameter. Further study is necessary, however, determine how widespread these changes are nd how important they may be clinically.

It has been shown only recently that changes haracteristic of hypertension occur in the cuneous blood vessels of patients who have essenal hypertension and that these changes are simir to those long known to be present in the rterioles of the ocular fundi, pectoral muscles, idneys and other structures and organs of the ody. These changes must be important clinically, nce the skin contains such a large part of the naller blood vessels in the body. In coarctation f the aorta, it has been noted that hypertensive ascular changes are present in the skin, being f equal degree in skin from the calf and from ne arm.

A group of diseases clinically different but havg somewhat similar histologic changes in the rterioles include acrocyanosis, livedo reticularis, hronic pernio and hypertensive ischemic ulcers f the leg. The predominant histologic changes re varying degrees of arteriolar spasm and scleros. The clinical picture is somewhat different the four diseases. Spontaneous nodular lesions nd ulcers may occur in the extremities in all of e conditions. The appearance of the early sion and its development suggest infarction of the skin, and the ulcer that is produced is of an ischemic type.

These diseases, like Raynaud's disease, occur predominantly in women. The explanation for this is not always clear. Chronic pernio (chronic chilblain) probably occurs more frequently in women because they do not protect their legs adequately from the cold. This also may be the explanation as to why complications are seen more often in women who have livedo reticularis than in men with the same condition. Hypertensive ischemic ulcer occurs mainly in women more than sixty years of age who have moderate to severe hypertensive vascular disease that has been present for many years. One explanation for the predominance of this condition in women is that fewer men than women with severe hypertensive disease live beyond sixty years of age.

Most noninflammatory diseases of the small blood vessels have their predominant clinical manifestations in the skin. When ulceration occurs from infarction, the initial lesion is similar in all. The first abnormality usually observed is a pink or red cutaneous plaque that becomes blue and purpuric within a week or ten days. In most instances, a hemorrhagic bleb develops on the surface of the plaque, and this may break down to form a typical superficial ischemic-appearing ulcer. The surface of the ulcer is usually sensitive and may be extremely painful, especially when it is not covered to prevent drying out of the region.

Acrocyanosis

Acrocyanosis is characterized by persistent blueness and coldness of the extremities, usually of the entire hands and feet. The condition is usually primary (idiopathic), although occasionally it may be seen as a secondary feature associated with signs of pulmonary osteoarthropathy. Trophic changes and gangrene do not occur; ulceration is rare and usually results from local trauma. Because of the pronounced blueness, many patients are concerned by the uncomely appearance and some fear gangrene.

Presented at the Symposium on Peripheral Vascular isease co-sponsored by the Minnesota Heart Association nd the Mayo Foundation, Rochester, Minnesota, Sepmber 24, 1957.

From the Section of Medicine, Mayo Clinic and ayo Foundation, Rochester, Minnesota. The Mayo oundation is a part of the Graduate School of the niversity of Minnesota.

Treatment.—The condition is usually innocuous and is more annoying than dangerous. In most cases, no treatment is indicated other than a discussion with the patient of the nature of the condition and of the fact that it is not dangerous and is not likely to give any severe disability. In an occasional instance in which the condition is severe, especially when excessive sweating is associated, sympathectomy may be advisable.

Livedo Reticularis

This disorder is characterized by a bluish, reddish, netlike pattern of discoloration that may involve all four extremities; occasionally it occurs over the entire body. The discoloration is permanent, although the intensity may vary with changes in environmental temperature. The color changes and minor discomfort are the only symptoms in about 90 per cent of the patients. The remaining 10 per cent may have disabling chronic ulceration, usually on the lower extremities and most commonly on the lower part of the leg.

Livedo reticularis may be primary and idiopathic or it may be symptomatic of other diseases, such as systemic lupus erythematosus and periarteritis nodosa. In the primary (idiopathic) type, the distribution of the color change is likely to be diffuse and symmetric, whereas the color changes in the symptomatic type are more disseminated and patchy. In most cases, livedo reticularis is not of serious significance. However, it is not rare. About 400 patients in whom this diagnosis was made were seen at the Mayo Clinic during the period between 1945 and 1955. In the symptomatic type, the nature of the underlying disease determines the prognosis.

Treatment.—In mild disease of the idiopathic type, the condition is more alarming than harmful, and reassurance and protection from cold are all that may be indicated. Sympathectomy occasionally may be advisable for those patients who are unable to protect themselves from the cold satisfactorily and who have distressing symptoms of coldness and discomfort when exposed to cold. Two patients at the clinic who had unusual degrees of disability from coldness and paresthesia associated with livedo reticularis that interfered seriously with their occupation obtained great symptomatic relief after sympathectomy. Relief has persisted in these patients for seven and twelve years, respectively.

When ulceration occurs, an initial period c rest in bed is advisable. Local applications of saturated solution of boric acid or of a 0.5 pe cent solution of aluminum subacetate may be c value in clearing up local inflammation in an around the ulcer. Fever therapy by means of th intravenous injection of typhoid vaccine or poly saccharides (pyromen) may be helpful in healin the ulceration. Use of drugs that block th sympathetic nervous system also may be of value My colleagues and I have used with some succes hexamethonium bromide or hexamethonium chlo ride given subcutaneously in increasing doses u to a maximum of 25 to 40 mg. two or thre times daily. We have seen only one patient wit livedo reticularis in whom amputation of a extremity was eventually necessary.

Chronic Pernio (Chronic Chilblain)

This is a condition that affects women usuall in the fourth or fifth decade of life. Chronic pernio is often unrecognized. The disease is characterized by erythematous ulcerating lesions in th lower extremities, particularly around the ankle and the lower part of the leg. During the earlie stages of the condition, the lesions appear at th onset of cool weather and disappear during th warmer summer months. After the conditio has been present for several years, the lesion may appear during the cool days of the summe season. The lesions characteristically go throug a cycle in which they develop over a period o two or three weeks and then heal over a perio of an additional two or three weeks. The ulcer heal spontaneously, leaving a pigmented an sometimes a depressed scar in the skin. Afte many recurring crops of lesions, the conditio becomes more chronic and extensive chroni changes may be present in the skin of the legs with pronounced induration and a low-grade inflammatory reaction that is more or less persistent.

Treatment.—The best treatment is prevention It is important to prevent recurring crops of lesions so as to forestall the chronic indurative stage of the disease in which treatment is extremely difficult and in which it may be impossible to clear up the lesions. Prevention can be accomplished usually by protection from cold and from injury. It may be necessary for the patient to move to a climate that is warmer during the fall and winter months, one in which the temperature is usually more than 80° F. and the air is dry. Lesions may develop in susceptible patients even though they live in regions ordinarily considered to have relatively mild fall and winter seasons if the temperature goes down during the winter to as low as 50 or 60° F.

When chronic lesions are present that do not clear up spontaneously, the use of vasodilating procedures or drugs may be helpful in eradicating the lesions. The value of sympathectomy in chronic pernio is uncertain. Several of our patients who have had chronic pernio associated with moderately severe hypertension and in whom sympathectomy was done for the hypertension had immediate disappearance of the lesions on the legs. However, recurrence of lesions during subsequent cool or cold weather was not entirely prevented in any of these patients after sympathectomy.

Hypertensive Ischemic Ulcer of the Leg

This is a relatively rare condition. About 100 patients with this condition have been seen at the clinic since it was first described here in 1941. It is usually an unrecognized clinical syndrome. The condition occurs predominantly in women between fifty and seventy years of age. The ulcers may begin spontaneously or may be induced by injury. The lesions most often are located low on the lateral and posterior surfaces of the leg. The ulcers usually are solitary, although injury may induce ulceration in several regions simultaneously. The typical ulcer apparently enlarges by extension of the purpuric hemorrhagic zone into the normal skin surrounding the ulcers. The diameter of the ulcers may range from 1 to 15 cm. The fully developed lesion is often rather painful. The patient may consult a physician more because of the severe pain and disability from pain in the ulcer than from concern about the condition itself. All lesions heal eventually but, if they are not treated, the healing may be extended over many months. In one of our patients, healing did not occur until after a fifteen-month period.

Treatment.—Prevention is also important in the treatment of hypertensive ischemic ulcers of the legs. It is much easier to prevent the initiation of the lesion than to treat it after it has appeared. In the program of prevention, special care of the feet and protection of the feet and legs are of utmost importance. The ankles and legs should be protected especially from minor injury, which is often an initiating factor in the appearance of the lesion.

When an ulcer already is present, it is best to start treatment by a short period of rest in bed, and it may be advisable to keep the patient relatively inactive at least until the ulcer has shown considerable evidence of healing. Appropriate sedative or narcotic drugs are often necessary for control of the severe pain. Local infection may be treated by a saturated solution of boric acid or a solution of antibiotic drugs. The hypertensive ischemic ulcer apparently does not tolerate application of ointments and salves well, and use of such agents should be avoided in most cases. Sympathectomy or the use of one of the sympathetic blocking drugs, such as is given in the treatment of hypertension, may be of help in hastening the healing of the lesion. When the ulcers are large and when the surface of the ulcer is in good condition, skin grafting also may shorten greatly the period of recovery and healing.

MEDICO-LEGAL FILMS AVAILABLE

Seventeen Questions and Answers have been prepared by the Law Department of the American Medical Association and The Wm. S. Merrell Company to supplement "The Medical Witness," a film on medical testimony.

The nine-page Question and Answer folder is available to any physician on request and should be of special interest to program chairman, medico-legal committees and the officers and staff of medical societies. For free copy, write to: Medical Witness Questions and Answers, The Wm. S. Merrell Company, Cincinnati 15, Ohio.

Information about the films, "The Medical Witness" and "The Doctor Defendant," will also be sent on request. These two films are in a series of medico-legal subjects now being filmed by Merrell in co-operation with The American Medical Association and The American Bar Association.

Raynaud's Disease

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FOR MANY YEARS following Raynaud's thesis on local asphyxia and symmetrical gangrene of the extremities,[1] the term "Raynaud's disease" was applied indiscriminately to a variety of conditions which, on close scrutiny and in the light of later knowledge, bore little or no resemblance to the author's original description. It is unfortunate that this early confusion in terminology has left a mistaken impression that persists even today, especially in lay circles where the name "Raynaud's disease" strikes terror and is equated to gangrene and amputation.

Hutchinson[2] was among the first to perceive that Raynaud was describing a symptom and not a single disease. It remained for Allen and Brown[3,4] to bring order out of chaos by carefully reviewing the literature on the subject and clearly defining Raynaud's phenomenon as a symptom of many diseases of which true or primary Raynaud's disease is but one. Just as "all that glitters is not gold," and "all that wheezes is not asthma," so all that blanches is not Raynaud's disease.

Definition of Terms

It is important, therefore, to begin this discussion with a definition of terms.

Raynaud's Phenomenon.—Raynaud's phenomenon results from intermittent episodes of constriction of small arteries or arterioles in the extremities. This is manifested clinically by intermittent changes in color of the skin of the fingers or toes or more rarely the ears, nose, and lips. When the spasm leads to complete occlusion of the affected arteries and arterioles, the skin becomes white. This is the pallor phase of Raynaud's phenomenon. When the spasm leads to incomplete occlusion of the affected arteries and arterioles, the skin becomes cyanotic; this is the cyanotic phase of Raynaud's phenomenon. The episodes of vasospasm may be followed by a period of reactive hyperemia during which the color of the skin becomes excessively red and warm; this is the rubor phase. Some patients with Raynaud's phenomenon may exhibit all three phases of color change. Others may exhibit only the cyanotic phase, and some experience only the pallor phase. The phase of reactive hyperemia or rubor is inconstant, and when it does occur, it follows the episodes of vasospasm. Each phase may last from a few seconds to a few minutes.

Raynaud's Disease.—When Raynaud's phenomenon is primary, that is, when it exists in the absence of diseases or conditions to which it may be secondary, it is called "Raynaud's disease."

This distinction between Raynaud's phenomenon which is a symptom and Raynaud's disease which is a distinct clinical entity is of utmost importance with respect to proper treatment and ultimate prognosis.

Pathophysiology

The pathophysiology underlying the Raynaud's phenomenon is poorly understood. That the intermittent episodes result from spasm of small arteries and arterioles is generally accepted. Whether this predilection to spasm is due to an inherent irritability in the walls of the involved vessel or is due to hyperexcitability of the sympathetic nervous system has not been determined. Organic occlusion of digital arteries does not occur in true Raynaud's disease, although it may be seen when Raynaud's phenomenon is secondary to other conditions. Larger arteries, such as the brachial and its branches, and the popliteal and its branches, are always open in true Raynaud's disease, and this is helpful to the clinician in that demonstration of occlusion of major arteries makes it unlikely that a patient with Raynaud's phenomenon has true Raynaud's disease.

Presented at the Symposium on Peripheral Vascular Disease co-sponsored by the Minnesota Heart Association and the Mayo Foundation, Rochester, Minnesota, September 24, 1957.

From the Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Diagnosis

In making a diagnosis of Raynaud's disease one must first establish that the patient has *bona fide* Raynaud's phenomenon, for although not all patients with Raynaud's phenomenon have Raynaud's disease, all patients with Raynaud's disease have Raynaud's phenomenon. A carefully taken history usually suffices to establish the presence of Raynaud's phenomenon, but in doubtful cases the vasospastic episodes can frequently be precipitated by having the patient grasp an ice cube or other cold object.

The second step in the diagnosis of Raynaud's disease is to rule out other causes for Raynaud's phenomenon. The diseases and conditions with which Raynaud's phenomenon may be associated are listed in Table I. Raynaud's phenomenon is sometimes the presenting manifestation or symptom of these conditions, and only a detailed history, careful physical examination, and indicated laboratory data will reveal the underlying cause for it. At times the primary disease may escape detection for several months during which Raynaud's phenomenon is the only symptom. Many potentially serious conditions, some of which might result in loss of limb or life, may cause Raynaud's phenomenon (Table I). It is easy to see, therefore, how early writers became confused in their concepts of Raynaud's disease and attributed major amputations and even death to it.

Allen and Brown[3,4] have set forth five criteria for making a diagnosis of true Raynaud's disease. These are (1) Raynaud's phenomenon excited by cold or emotion; (2) bilateral Raynaud's phenomenon; (3) ischemic lesions, when present, limited to small areas of cutaneous gangrene; (4) exclusion of diseases listed in Table I that might be causal, and (5) symptoms for at least two years. Experience has shown that the diagnosis of Raynaud's disease established by adhering to these criteria will be accurate in 95 per cent of cases.[5]

Clinical Characteristics

Raynaud's disease occurs primarily in young women. At the Mayo Clinic 77 per cent of patients with Raynaud's disease are women, and of these women, 78 per cent are less than forty years of age at the time of onset of their disease.[5,6]

The majority of patients state that exposure to cold is the usual stimulus for precipitating Raynaud's phenomenon. Approximately 20 per cent also notice that Raynaud's phenomenon appears under situations of emotional stress, even though the environmental temperature is warm.

TABLE I. CONDITIONS AND DISEASES TO WHICH RAYNAUD'S PHENOMENON MAY BE SECONDARY

(These must be excluded before a diagnosis of primary Raynaud's disease can be made)

- I. Occlusive arterial disease
  - A. Arteriosclerosis obliterans
  - B. Thromboangiitis obliterans (Buerger's disease)
- II. Systemic diseases
  - A. Scleroderma
  - B. Disseminated lupus erythematosus
  - C. Periarteritis nodosa
  - D. Rheumatoid arthritis
- III. Abnormalities of blood
  - A. Cryoglobulins
  - B. Cold agglutinins
- IV. Intoxication from:
  - A. Lead
  - B. Arsenic
  - C. Ergot
- V. Neurogenic lesions
  - A. Diseases of nervous system
  - B. Thoracic outlet compression (scalenus anticus and hyperabduction syndromes)
- VI. Trauma
  - A. Occupational
    - 1. Raynaud's phenomenon in typists and pianists
    - 2. Occupational occlusive arterial diseases of hand
    - 3. Pneumatic-hammer disease
  - B. Late result of cold injury
    - 1. Trench foot
    - 2. Immersion foot
    - 3. Frostbite

Nearly all patients with Raynaud's disease first notice the vasospastic phenomenon in their fingers. Approximately 40 per cent also will have symptoms in their toes. It is unusual for symptoms to appear first in the toes, and even more unusual for symptoms to remain confined to the toes in true Raynaud's disease. Occasionally the vasospastic phenomena of true Raynaud's disease may be unilateral for several years before they become bilateral. However, when unilateral Raynaud's phenomenon is present. one should suspect some local cause, such as occlusive arterial disease, thoracic outlet syndrome, or chronic occupational trauma with occlusion of the palmar arterial arch. Approximately 5 per cent of women with Raynaud's disease will have a family history of Raynaud's phenomenon.

Approximately 10 per cent of women with Raynaud's disease have hypertension (with blood pressure of more than 150 mm. of mercury systolic and 90 mm. diastolic), and approximately 15 per cent have migraine headache. The frequency of functional complaints among women with Raynaud's disease has led Mufson[7] to postulate that in some cases, at least, psychosomatic factors play a prominent role in etiology.

Physical Examination

Complete physical examination is essential in the search for diseases or conditions to which the Raynaud's phenomenon may be secondary.

TABLE II. COMPLICATIONS OF RAYNAUD'S DISEASE AT OR BEFORE FIRST VISIT TO CLINIC FOR 474 WOMEN[5]

Complication	Women	Per Cent
Trophic changes	40	8
Sclerodactylia	34	7
Trophic changes and sclerodactylia	23	5
Calcinosis cutis	1	0.2
Amputation of phalanges	2	0.4

The extremities of patients with uncomplicated Raynaud's disease usually appear normal between vasospastic episodes. Sometimes the hands or feet or both tend to be cool and perspire excessively. Arterial pulsations are palpable at the wrist and in the feet, although an unusually warm environment occasionally is necessary to make them evident.

Special Methods of Investigation

Special procedures such as skin temperature studies, oscillometry, and plethysmography are of little practical value in making the diagnosis of Raynaud's disease. Arteriography is sometimes helpful in ruling out occlusive arterial disease as a cause for Raynaud's phenomenon.

Complications of Raynaud's Disease

Complications of Raynaud's disease occur in less than 30 per cent of cases and are usually not severe. They consist of trophic changes, sclerodactylia, and calcinosis cutis. They may appear separately or together.

Trophic changes occur in the tips of digits and around the nails and include chronic fissures, ulcers, superficial infarcts in the skin, and chronic paronychia. These lesions may heal leaving pitted scars. Extensive gangrene does not occur as a complication of Raynaud's disease.

Sclerodactylia consists of sclerodermatous changes confined to the skin of the fingers. As a complication of Raynaud's disease it never involves more proximal parts and, therefore, can be distinguished from the more extensive involvement seen in diffuse scleroderma and acrosclerosis.

Calcinosis cutis occasionally occurs at the tips of digits of patients who have Raynaud's disease. It appears as tiny globular deposits which may ulcerate and discharge their contents.

The incidence of these complications is not great (Table II). Furthermore, the disability arising from the complications is often minimal. It is surprising, though gratifying, that complications led to amputations for only two (0.4 per cent) of 474 women before or at the first visit to the Mayo Clinic for Raynaud's disease.[5] These amputations were minor, involving only one or more phalanges and were performed for painful and nonhealing ulcers. No major amputations were necessary.

Prognosis in Raynaud's Disease

A recent study has shown that the prognosis for patients with true Raynaud's disease is surprisingly good.[5] Follow-up studies were made on a series of 307 women from three to forty-six years (average seventeen years) after onset of symptoms of Raynaud's disease. None of these women had undergone sympathectomy. Most of them were carefully instructed about protection of their hands from cold and trauma, but otherwise treatment was not standardized, and most patients had not followed any particular therapeutic regimen for prolonged periods.

Ten per cent of the women reported that Raynaud's phenomenon disappeared during the follow-up period; 36 per cent reported that Raynaud's phenomenon had improved. Only 16 per cent reported that Raynaud's phenomenon was worse at the time of follow-up. The remainder noted no particular change in the severity of Raynaud's phenomenon. It is interesting that a detailed study of the smoking habits of these women with Raynaud's disease failed to produce evidence that cigaret smoking had any effect, adverse or otherwise, on the course of Raynaud's phenomenon.

The complications of Raynaud's disease frequently became less troublesome or disappeared entirely. Eighty-seven per cent of the women who had trophic changes of their digits initially had no further ulcerations in the period of follow-up. Sclerodactylia was less noticeable or was causing no disability for 70 per cent of the women who had it initially.

Only 8 per cent of women who were free of the complications of Raynaud's disease when the diagnosis was first made subsequently had trophic changes, sclerodactylia or calcinosis cutis, either singly or in various combinations.

No amputations were necessary for the complications of Raynaud's disease during the period of follow-up.

Twelve (4 per cent) of the 307 women in this group died before the end of the follow-up period. None of the deaths could be attributed to Raynaud's disease or its complications. Indeed, there s every reason to believe that Raynaud's disease s compatible with a normal life span. But this is not true for secondary Raynaud's phenomenon which may be associated with serious and lethal diseases. For instance, 38 per cent of 98 women with secondary Raynaud's phenomenon who were traced died during a similar follow-up period.[5] The average age at time of death for the women with secondary Raynaud's phenomenon was forty-seven years and 40 per cent were less than forty years of age when they died.

Treatment

Reassurance is the first step in treatment of Raynaud's disease. The physician should first reassure himself that his patient has true Raynaud's disease and that the Raynaud's phenomenon is not secondary to some other disease. He then can confidently reassure his patient and allay the apprehension that arises from misinformation about the nature and prognosis of Raynaud's disease. The patient should be told that major amputation is never necessary for Raynaud's disease and that minor amputations are rarely required.

Protection of the hands and feet from cold and mechanical trauma is important. Cold objects should not be handled without the use of gloves; thus patients with this disease should wear gloves when handling items in the refrigerator or freezer. For going out of doors in cold weather, patients with Raynaud's disease should wear suitable clothing to keep the body as well as the extremities warm. Prolonged immersion of the hands in water as in washing dishes can be harmful, especially when irritating soaps or detergents are used.

Vasodilating drugs such as tolazoline (priscoline) and dibenzyline, or ganglion blocking drugs, such as hexamethonium or mecamylamine (inversine), are sometimes helpful in reducing the frequency of vasospastic episodes. Often, however, such medication is not needed, and in mild cases the side effects which they produce will be more troublesome than the Raynaud's disease. An ointment which contains 2 per cent glyceryl trinitrate applied locally to the involved extremities has proved helpful in some cases.

Surgical sympathectomy should be reserved for those patients whose Raynaud's disease is severe and progressive in spite of adequate medical management. In the experience of my colleagues and me, sympathectomy is necessary for less than 20 per cent of patients with Raynaud's disease.

TABLE III. RESULTS OF SYMPATHECTOMY FOR 70 WOMEN WITH RAYNAUD'S DISEASE[8]

Result	Upper Extremities Denervated		Lower Extremities Denervated	
	Patients	Per Cent	Patients	Per Cent
Excellent	7	10	15	83
Good	30	44	2	11
Fair	6	9	0	
Poor	25	37	1	6
Total	68		18	

Data derived from follow-up study of seventy women with Raynaud's disease who had various types of operations to interrupt the sympathetic nervous pathways prior to 1945 are presented in Table III.[8] The average period of follow-up after operation was twelve years, and in one case it was twenty-eight years. Two patients had operations for the lower extremities only, fifty-two for the upper extremities only, and sixteen, for both the upper and lower extremities.

The results of sympathectomy are consistently better in the lower than the upper extremities, and this striking difference has been reported by other authors.[9-13] All too frequently, sympathectomy for the upper extremities will produce good or excellent initial results, only to be followed by relapse within a few months. The preganglionic thoracic sympathectomy described independently by Smithwick[14] and Telford[15] and the postganglionic cervicothoracic sympathectomy described by Adson[16] produced similar results. There was no operative mortality.

None of these patients had only second thoracic ganglionectomy, and none had the more extensive procedures advocated recently by Ray.[17]

Lest the reader gain the impression that the results of surgical treatment of Raynaud's disease in the upper extremities is little better than conservative treatment, it should be emphasized that only progressive and recalcitrant cases were chosen for such treatment. In other words, most of these patients had failed to respond to medical treatment.

Follow-up studies were conducted also on a group of fifty-four women who had sympathectomy for Raynaud's phenomenon secondary to acrosclerosis, rheumatoid arthritis, occlusive arterial disease, periarteritis nodosa, and other diseases. The results were much worse than in the group with Raynaud's disease. In this series only 20 per cent of women obtained good or excellent benefit from sympathectomy for the upper extremities, and only

50 per cent obtained good or excellent benefit from sympathectomy for the lower extremities. This is another reason why it is so important to differentiate between true Raynaud's disease and secondary Raynaud's phenomenon.

Summary

Raynaud's phenomenon results from intermittent spasm of small arteries and arterioles, usually in the fingers, less often in the toes. It can be associated with and secondary to many unrelated diseases and conditions some of which are serious and even fatal. The prognosis is therefore uncertain until a diagnosis of primary or true Raynaud's disease can be substantiated by ruling out other causes for Raynaud's phenomenon. Raynaud's disease is a benign condition; no mortality is associated with it, and serious disability infrequently results. Raynaud's disease does not lead to extensive gangrene, and major amputations are never necessary. Raynaud's disease is the most common but least serious of the many conditions with which Raynaud's phenomenon may be associated.

Conservative treatment is effective for most patients with Raynaud's disease, but in resistant cases surgical sympathectomy is indicated.

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STORY OF CEREBRAL PALSY

In a booklet just published by the National Society for Crippled Children and Adults, Earl Miers, prominent author, editor and Civil War authority, has written his own story of conquest of cerebral palsy in "Why Did This Have to Happen?"

To assist in solving the perplexing problems which confront parents in raising handicapped children, Mr. Miers offers inspiration. In his moving life story, he points his lesson thus: "I watch two men coming down the street. One propels himself on crutches, the other strides along on a sound pair of legs . . . My eyes cannot tell me which of these two is more severely handicapped . . . I speculate over which is fitter to survive in the specific environment that we share. Which is the more courteous? The more likely to smile? The more aglow with a warmth for life? The more responsive to another's need? The more willing to render faithful service to his employer, his community, his family? Which is better trained in his work and thus more capable of bringing insight and imagination to his labors? Which is less filled with prejudice, less consumed with deceit?"

He goes on to say: "Judge and musician, innkeeper and statesman, actor and surgeon—each endures by holding onto beliefs. For any person possessing firm foundations of intellectual strength and spiritual stamina, legs that wobble or arms that shake constitute inconsequential handicaps. As long as any man has a right to be born, a purpose to fill on earth, then a crippled child holds the same right, the same purpose."

The booklet, which is one of a series designed to help parents of crippled children, is available at the nominal charge of 25 cents from the National Society for Crippled Children and Adults, 11 South La Salle Street, Chicago 3, Illinois.

Periarteritis Nodosa and Temporal Arteritis

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PERIARTERITIS NODOSA is a disseminated disease of unknown origin characterized by focal inflammatory lesions which involve chiefly medium-size and small arteries and arterioles. It has been classified as a so-called collagen disease, and as such takes its place with other rheumatic and "pararheumatic" syndromes as an inflammatory and degenerative disease of the connective-tissue system.

Incidence.—The incidence of this disease is somewhat higher among males than among females. It can occur at all ages, but a majority of the patients are between the ages of twenty and forty years.

Etiologic Aspects.—As is true of all obscure conditions, many opinions as to cause have been advanced but none has been satisfactorily confirmed or completely accepted.

Present opinion is that multiple etiologic factors may be involved in the production of the disease, with a sensitivity reaction to either drugs or infectious processes playing a prominent role.

Pathologic Aspects.—The pathologic pattern consists of a primary injury to the wall of the vessel, with necrosis of the media, destruction of the internal elastic membrane and infiltration of the adventitia by polymorphonuclear leukocytes, many of which may be eosinophils. Aneurysms occur in probably less than 20 per cent of cases. Occasionally these may rupture. When the process extends to the intima, the smaller vessels frequently become occluded. Characteristically, arterial lesions in any and all stages of activity are present at the same time. At necropsy, rarely, all the lesions are healed.

Symptoms and Signs.—The symptoms and signs are protean. They may vary from time to time and this variation may produce a most confusing clinical picture. They depend entirely upon the stage of the disease and the organs or tissues involved. In general, periarteritis nodosa creates the impression of an infectious disease with toxemia. Frequently, it is characterized by cachexia associated with malaise, anorexia and low-grade fever, with additional symptoms and signs referable to the organs and tissues affected.

The renal manifestations usually are those of nephrosclerosis or glomerulonephritis. Hypertension is a frequent finding, and usually it is associated with vascular and other changes in the kidneys. At times, it is difficult to distinguish periarteritis nodosa from malignant hypertension.

Many of the patients die in uremia, but the only signs of renal involvement may be microhematuria or albuminuria.

Abdominal pain is one of the common early symptoms. Hepatomegaly and jaundice may occur, but usually are not pronounced. Complications, such as perforation of a gastric or intestinal ulcer, infarction of the bowel or necrosis of the pancreas, may develop and the diagnosis may be established at exploratory laparotomy.

Tachycardia is common. Electrocardiographic changes usually are nonspecific and variable. "Strain" patterns may be noted in patients with hypertension. Although anginal pain is uncommon, myocardial infarctions often are found at necropsy. Pericarditis may occur, and cardiac failure is a common terminal manifestation of the disease.

The most common pulmonary manifestations are wheezing, dyspnea and cough. These symptoms may be secondary to either asthma or heart failure. The incidence of asthma in periarteritis nodosa probably is less than 25 per cent. Hemoptysis, due to infarction or rupture of an involved vessel, may occur. Pleuritis with pleural effusion secondary to pneumonitis or infarction is not uncommon. The roentgenogram of the thorax may

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show no specific changes. A fanlike pulmonary infiltration extending out from the hilar region, although not pathognomonic of this disease, may suggest the diagnosis in an otherwise obscure problem. Loeffler's syndrome may be a variant of the disease, confined to the lungs, from which recovery occurs, in most instances. Terminal bronchopneumonia frequently is the immediate cause of death.

We have noted overt clinical evidence of involvement of peripheral nerves in approximately 50 per cent of our cases. As a rule, such involvement is of the mononeuritis multiplex type, with asymmetric involvement of single nerves. Scotomas, blurring of vision and paresis of extraocular muscles may occur. The ocular fundi may exhibit typical vascular lesions in the choroid and retina, retinal detachment or hypertensive retinopathy. Cerebral hemorrhage as the result of hypertension, infarction or rupture of an involved vessel is an occasional cause of death.

Symptoms in the joints are noted frequently; they may vary from transitory arthralgia to migratory arthritis. Muscular pain and soreness are common. Osteoporosis may occur. We have encountered aseptic necrosis of the hips in one of our cases. The coexistence of periarteritis nodosa with other rheumatic and "pararheumatic" diseases has been observed by us and by others.

The endocrine organs most often involved are the adrenal glands, pancreas and testes.

Many types of lesions of the skin have been described, including erythema, scarlatiniforme, petechial, purpuric, maculopapular, urticarial and vesicular eruptions, livedo reticularis, subcutaneous nodules and cutaneous infarction with necrosis and ulceration. A special cutaneous form of the disease has been described as "periarteritis nodosa cutis." This usually is unaccompanied by systemic manifestations, and appears to have a benign course.

Laboratory Aspects.—Laboratory findings for the most part are nonspecific. Moderate to marked leukocytosis may be present. Eosinophilia is inconstant; it occurs in less than 20 per cent of cases. The erythocyte sedimentation rate invariably is rapid during the active phase of the disease; values of more than 100 mm. in 1 hour (Westergren) are not uncommon. Secondary anemia often is present; it may be mild to severe. The association of acquired hemolytic anemia and platelet thrombosis with periarteritis nodosa has been reported. Biologic falsely positive serologic reactions to tests for syphilis and increased values for plasma globulin may be obtained. Porphyrinuria has been noted when the liver is involved. Hematuria, albuminuria and cylindruria are common.

Clinical Diagnosis.—The disease should be suspected in a patient who has an obscure type of illness characterized by fever, progressive decline of general health, and the symptoms and signs of a diffuse systemic process, with involvement of several organs and tissues, particularly the kidneys, gastrointestinal tract and peripheral nerves. The presence of a rapid sedimentation rate, leukocytosis with or without eosinophilia, asthma and hypertension, and failure of the condition to respond to conventional treatment make the diagnosis even more likely. In the final analysis, however, a definite diagnosis can be established only by histologic examination. A positive result of biopsy is the best diagnostic evidence of the disease, but a negative result does not exclude the diagnosis.

Prognosis.—In general the prognosis is poor. However, a few spontaneous recoveries have been reported. Spontaneous recovery may be expected under the following circumstances: confinement of the disease predominantly to nonvital structures, minimal involvement of organs followed by healing, and localization of the disease to part of a single organ.

Treatment.—Until the introduction of cortisone and related steroids, there was no specific treatment which would either prevent or alter the course of periarteritis nodosa.

We have administered cortisone or related steroids to approximately fifty patients with periarteritis nodosa. Follow-up data have been obtained in thirty cases in which cortisone or corticotropin was used. Clinical diagnoses were confirmed by biopsy in all cases. Doses of each hormone were initiated at fairly high levels, and were regulated according to the patient's response. In general, 300 mg. of cortisone was administered on the first day and 150 to 200 mg. was given daily thereafter for six weeks. The dose was then gradually altered by decrements of 12.5 to 25 mg. every three or four days, and treatment was discontinued in most instances after three to four weeks. We prefer the intramuscular route of administration of cortisone, at least during the initial six weeks of treatment. Our clinical impression has been that the results are more favorable and the incidence of relapses after cessation of therapy is less when the intramuscular route is employed than when the oral route is used. The reason for this is not clear, but it may be associated with a greater cumulative effect of the drug obtained by the intramuscular route as opposed to the oral route. The dose of corticotropin was one-third to one-half that of cortisone.

Additional measures included restriction of sodium in the diet and the administration of potassium chloride in doses of 3 to 6 gm. a day to patients with adequate renal function. Some patients required more than one course of treatment, and in a few instances it was necessary to continue suppressive therapy with orally administered cortisone in doses of 25 to 75 mg. daily.

Clinical phenomena that followed the administration of cortisone and corticotropin in these cases include: (1) prompt subjective relief, (2) subsidence of fever in most cases within twenty-four to seventy-two hours, (3) gradual decrease of sedimentation rates to normal, (4) partial relapses among some patients after withdrawal of the use of the hormones, and (5) improvement in these patients after treatment was resumed. Despite initial improvement, six patients died shortly after treatment was instituted or soon after administration of the hormones was discontinued. In two of these cases necropsy revealed evidence of complete healing of all arterial lesions. In the process of healing, fibrous obliteration of the lumens of the involved vessels had occurred, resulting in widespread visceral infarction.

In the other four cases, necropsy showed widely disseminated arterial lesions in all stages of activity, with multiple visceral infarctions. Three other patients died—four, twenty and thirty months, respectively, after treatment. These patients died elsewhere, and postmortem examinations were not performed. Although adequate information was unobtainable, death was presumed to be the result of irreversible effects of the disease. The disease of the remaining twenty-one patients appeared to be either in remission or adequately suppressed when they were last observed after intervals ranging from one year to five years after the initial course of treatment. However, in most instances, evidence of some residual vascular damage had persisted.

Conclusions.—It appears that cortisone and corticotropin can induce clinical remissions *in* or suppress activity *of* periarteritis nodosa in some cases, and that they may alter effectively the primary pathologic process. Treatment should be instituted early, before widespread vascular damage has occurred, and should be intensive and prolonged. The suppressive effects of prednisone and prednisolone appear to be comparable to those of cortisone administered by the oral route.

Temporal Arteritis

Temporal (cranial) arteritis is a clinical and pathologic entity that occurs in elderly persons. It is characterized by symptoms and signs referable to inflammation of the temporal arteries, by constitutional effects suggestive of a low-grade subacute or chronic infectious process and by histologic evidence of a granulomatous arteritis of the affected vessels.

Etiologic Aspects.—The cause of temporal arteritis is unknown.

Incidence.—In practically all the reported cases the patients have been more than fifty-five years old, and most of them have been more than sixty-five years old. The incidence according to sex is about equal.

Pathologic Aspects.—The disease affects isolated segments of the temporal arteries and occasionally other arteries, especially branches of the carotid arteries. Grossly, the affected arteries are larger than normal, and may be nodular. Frequently they appear to be solid cords with little or no lumens. Histologically, there is usually narrowing or complete occlusion of the lumen produced by intimal proliferation, with or without thrombosis. There are definite but rather localized periarteritis and mesarteritis. Generally, one sees focal necrosis of the media and replacement with gramulomatous tissue containing numerous foreign-body giant cells. Eosinophilic infiltration is uncommon and aneurysmal dilatations are rare.

Symptoms and Signs.—The onset of the disease may be insidious or sudden, and the course is that of a subacute or chronic infection, with local and systemic manifestations. Local manifestations include severe, throbbing headache with tenderness, swelling and induration of the temporal and occasionally the occipital arteries. Often there are erythema of the overlying skin and pain in the adjacent structures, such as the scalp, face, eyes and jaws. The lesions may begin on one side of the head, but as a rule the arteries on both sides ultimately become involved. Early in the course of the disease, the affected arteries may pulsate, but later the pulsations may disappear.

Generally, a systemic reaction is present, with low-grade fever, malaise, muscular aching, anorexia and loss of weight. These may occur a few weeks or even a few months before the local signs become apparent.

Laboratory Aspects.—Laboratory findings are constant but nonspecific. Mild anemia and mild to moderate leukocytosis usually are present. The sedimentation rate is invariably rapid during the active phase of the disease, generally exceeding 100 mm. in 1 hour; normal or only slightly increased values make the diagnosis impossible.

Clinical Diagnosis.—Ocular involvement occurs in more than 50 per cent of the cases. Involvement of the eyes usually is due to occlusion of the central retinal artery or branch arterioles or to ischemic optic neuritis, and it may result in partial or complete loss of vision in one eye or both eyes. The eyes may become involved within a few days or as late as several months after the onset of the disease. In general, however, ocular symptoms begin within three to six weeks after the onset of headache and after a somewhat shorter interval following the appearance of induration and tenderness of the temporal arteries.

Loss of vision may occur suddenly or slowly over a period of several weeks. The symptoms may vary from blurring of vision and different patterns of defects in the visual fields to complete blindness. More than a third of the patients whose eyes are involved lose their sight permanently, and only a few regain normal vision. Paresis of extraocular muscles may occur. We believe that this paralysis is caused by ischemia of the extraocular muscles resulting from involvement of the muscular branches of the ophthalmic artery, and we think it may be transient.

Cerebral complications have been reported, but are uncommon. Sudden complete deafness has been noted, and in one of our cases the radial arteries were affected by the same pathologic process as the temporal arteries.

As a rule, it is not difficult to make the diagnosis if the possibility of temporal arteritis is kept in mind whenever an elderly patient complains of pain and soreness in the temporal region. We know of no other condition in which localized tenderness and induration are felt along the course of the temporal arteries. An elderly patient who has transitory amblyopia or sudden loss of vision should be carefully examined for tender, indurated or pulseless temporal or occipital arteries, and the symptoms should be reviewed from the standpoint of possible temporal arteritis. A rapid sedimentation rate invariably is present during the active phase of the disease. When the diagnosis is doubtful, it may be confirmed by biopsy of what appears to be an involved vessel.

Generally, the disease runs a self-limited course, lasting from two to thirty months, with an average duration of about six months. The active and symptomatic phase gradually subsides, although occlusion of the involved arteries may persist. Relapses may occur but are uncommon.

Although temporal arteritis usually is a benign and self-limited disease, a mortality rate of 12.5 per cent has been reported, deaths having been attributed to cerebral thromboses and disseminated arteritis, histologically similar to that in the temporal arteries. This is contrary to our observations in a comparable series of cases in which there were no deaths.

Treatment.—Until the advent of cortisone and related steroids the treatment of temporal arteritis was mainly symptomatic, consisting of the usual measures to relieve pain. Excision of a portion of the involved segment of the artery generally relieves the pain of local origin but seldom, if ever, has any effect on the general manifestations or the course of the disease. Roentgen-ray irradiation of the affected arteries and the periarterial injection of procaine hydrochloride likewise may relieve the local distress.

We have administered cortisone or related steroids to approximately seventy-five patients with temporal arteritis. Clinical diagnoses were confirmed by biopsy in most cases. We have recently reviewed fifty-five cases in which the diagnosis was proved by biopsy; we did this to evaluate the effect of steroid therapy. In general, the dose schedules were essentially the same as for patients with periarteritis nodosa. There appears to be little advantage in the parenteral over the oral route of administration of cortisone in this disease. In some instances, it was necessary to continue suppressive treatment with orally administered cortisone in doses of 25 to 75 mg. daily until the disease had completed its natural course.

Since we believe that in respects other than ocular, temporal arteritis is primarily a self-limited and benign condition, prevention and treatment of the associated visual loss are of paramount importance. All but a very few patients in this series were promptly relieved of their other symptoms by the administration of adrenal corticosteroid.

Thirty-one (56 per cent) of the fifty-five patients, at the time of admission to the clinic, had ocular involvement which was considered to be part of the syndrome of temporal arteritis. Twenty patients (36 per cent of the group) had some loss of vision at the time of admission, and one patient had transient amblyopia after the institution of hormonal therapy. In this group of twenty patients, six had unilateral blindness and five had bilateral blindness. The condition of none of the blind eyes improved under treatment. However, bilateral blindness did not develop in any patient during treatment. The percentage of occurrence of bilateral blindness increased from six on admission to seventeen at the time of discharge in a previous group of fifty-three patients with temporal arteritis seen at the clinic prior to the introduction of steroid therapy.

Total blindness and long-standing partial loss of vision are unlikely to abate with any type of treatment. When loss of vision is partial and of recent onset, some restoration of vision may occur, with or without treatment.

That the hormones promote histologic healing is subject to question, since follow-up biopsies in several of these cases revealed little more healing of the arterial lesions than would be expected in the natural course of the disease.

In the past we have not routinely administered anticoagulant agents to patients with temporal arteritis. We feel, however, that the combined use of anticoagulants and adrenal corticosteroids might be indicated and might be of value in certain instances, notably for patients who have had recent impairment or loss of vision in one eye. It is conceivable that in such cases inflammatory changes already may have developed in the ophthalmic artery of the presumably uninvolved eye, and that subsequent occlusion with loss of vision may supervene, with or without steroid therapy. The intermittent inhalation of 100 per cent oxygen has seemed to be of some benefit in cases of acute or recent visual impairment, but we have not been impressed with the value of vasodilating drugs or stellate-ganglion blocks in such cases.

Conclusions.—The chief value of adrenal corticosteroids in the treatment of temporal arteritis is to suppress the arteritis as long as it endures, and thus to safeguard the degree of vision which remains.

THERAPY OF TOYS IN CHILD'S PLAY

Dr. Grace Langdon, one of the country's well-known leaders in the field of child development and specialist in the relation of toys to everyday living, has written a booklet entitled "Your Child's Play," which was recently released as one of a series of booklets to be published by the National Society for Crippled Children and Adults as aids to parents of crippled children.

Dr. Langdon in her booklet points out that all children, whether crippled or not, live through play, and she offers guidance in play interests, materials, and facilities as well as friends with which to share them.

She says, "Play is much more than filling in time when a child has nothing to do. Through play a child investigates, explores, experiments, discovers. He learns the feel of things. He discovers what he can do with them. . . . He notices the motion of things around him, and with hands, arms, legs, joins in this movement through feeling its rhythm. . . . As a child plays he put his ideas and feelings into action. He finds out what will work and what will not. He picks up new ideas and tries them out. He enjoys, thinks, feels, lives through his play."

Twenty-two additional booklets, two more scheduled for publication in 1958, will comprise the Parent Education series as it is presently planned. These will be on subjects such as the young child in the family, orthopedic handicapipng conditions, problems of adolescence, and emotional growth in parents.

Purposes of the series, as set forth by Dean W. Roberts, M.D., executive director of the National Society, are to offer guidance to parents in creating an abundant and satisfying life experience for themselves and their children; to dispel feelings of loneliness and helplessness in facing problems of growth and development; to provide a medium through which the parent may grow with his child, and to give insight through competent information.

The booklets are available at the nominal charge of 25 cents each from the National Society for Crippled Children and Adults, 11 South LaSalle Street, Chicago 3, Illinois.

Peripheral Vascular Diseases

Evaluation of Treatment

EDGAR V. ALLEN, M.D.
Rochester, Minnesota

THIS PRESENTATION should more appropriately be entitled "An Excursion into Skepticism," for one needs much critical evaluation to keep an even scientific keel in a world in which claims for the value of various treatments are so common and evidence for such value is so sparse. The way of the skeptic is hard. Popularity contests are not won by seekers after truth. Yet skepticism, I believe, needs no defense, whereas credulity simply has no defense.

The history of all fields of human endeavor indicates that progress can be made only when people are dissatisfied with the current situation. Almost all research programs are predicated on beliefs that what is commonly accepted is false or inadequate. Inasmuch as this is the three hundredth anniversary of the death of William Harvey, who was one of the greatest proponents of objectivity in medicine, I may appropriately quote a small part from his perceptive writings. He stated: "I learned and taught anatomy not from books but from dissections, not from the position of philosophers but from the fabric of nature."

Criticism of Present Handling of Pharmaceuticals

To each of you almost every day comes printed material from pharmaceutical firms, which, if entirely true, would simplify the practice of medicine and improve the mental and physical health of almost everyone who is ill. I have been told that one pharmaceutical firm makes several hundred products which are marketed largely because physicians prescribe them or because people buy them without prescription. A physician has reported that he receives more than 2,000 pieces of advertising literature annually. The *Physicians' Desk Reference* for 1958 devotes 138 pages to listing about 27,000 "therapeutic items." The yearly bill for drugs includes these production and promotion costs, and profits for manufacturer and distributor as well. It is enormous, and I am sure that some of the money could be spent in a much better cause—for example, research.

If I have spoken critically of pharmaceutical firms, I have done so with the full knowledge that some are interested more in profit than in honesty. They must, of course, operate within existing laws, but such laws are woefully inadequate. I shudder every time I see the advertisements on television for such products as bufferin and anacin, and I remain eternally curious that such exaggerated claims are permitted. Although the fault lies chiefly with inadequate control, it is well for all of us to know that some of the shabby and dishonest presentation rubs off on physicians because the public cannot differentiate between the medical profession and a pharmaceutical firm. I am aware that many pharmaceutical firms have contributed greatly to scientific advancement by way of better drugs, but I lament the dilution of the virtues of many with the faults which proceed from the desire for great profit on the part of some.

Still I cannot leave the subject with a simple indictment of pharmaceutical firms. I have implied already that ordinarily a medicine cannot be marketed satisfactorily without the co-operation of many physicians who exhibit an amazing credulity. There seems to be common a naive belief that whatever is written is true. And for another reason I cannot restrict my criticism to pharmaceutical firms alone: in many instances the allegations as to the benefits of a preparation are based on studies reported by physicians who have no official association with pharmaceutical firms. I do not know the complete explanation for this situation, but it appears to represent lack of knowledge of the requisites for a scientific study on one hand and a desire to publish positive

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Presented at the Symposium on Peripheral Vascular Disease co-sponsored by the Minnesota Heart Association and the Mayo Foundation, Rochester, Minnesota, September 24, 1957.

From the Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

results on the other hand. There seems to be much more credit in establishing virtues for a product than in determining that a preparation has no virtue.

I feel compelled to say a few words concerning the attitude of editors of medical journals. They may publish articles from those of us who are not particularly skilled in consideration of this subject; too often they do not refer such articles to experts for evaluation. Moreover, some medical journals accept advertisements with little or no regard for the virtue of the product which they advertise. It is my firm belief that the editors of medical journals should accept advertising only for products which they know have virtue.

Underlying the total problem which I have discussed briefly is the desire to assert the positive view, the desire to believe the positive assertion, which sways editors, physicians, and people at large. Hardly any patient wishes to disappoint his physician with an unfavorable report. This situation can be handled best by the "double blind" test in which neither the patient nor the physician knows whether the patient is receiving a medicine or a placebo. The general lack of favor for this approach is an indication that physicians do not care to take trouble in finding the truth or that indeed they do not want to know the truth.

Over a period of years I have been asked to test this and that. When I have agreed, I have frequently been disappointed, for the results of study have usually indicated that a preparation had no virtue or much less virtue than I had anticipated on the basis of prior information.

Recommendations.—I know that one should never be wholly destructive in criticism. Because of this and the seriousness of the problem, I wish to make suggestions for an aggressive approach to the solution of it.

1. All pharmaceutical firms should voluntarily abstain from exaggerated advertising. (Such firms, of course, do not now adhere to the same policy in this regard.) I have little or no hope that this gratifying change will be accomplished, although any pharmaceutical firm which carried out such a policy would certainly endear itself to many physicians. There is, however, the problem of dividends and capital gains.

2. There should be control of advertising to restrict it to just and valid claims. Here again I see little reason for optimism. What would become of our television programs if people knew that bufferin is only aspirin plus a couple of antacids, and that anacin is only acetophenetidin with aspirin and caffeine added? In my opinion much of the television programming is of such a low order that I hope many of them will disappear. Unfortunately people might be forced into conversation or reading, both of which appear to be becoming increasingly painful.

3. For a period of one year, physicians without the ability of perceptive evaluation should throw all advertisements into the waste basket and refuse to converse with "detail men." There is a little danger in such a program, which could be avoided if a medical periodical such as the *Journal of the American Medical Association* would publish immediately any important discovery. In this third recommendation, I am wholly pessimistic; the pictures are so pretty!

4. Medical editors should refuse to publish articles which do not have the approval of experts. This actually is possible, although I fear many experts are too busy. One of my colleagues has mentioned "freedom of the press" in this regard. If this is an issue, and unprepossessing papers must be taken, then the editors should encourage publication of rebuttals.

5. Medical editors should accept advertising of only those products which have proved value. My pessimism deepens; medical journals would undoubtedly be more costly and physicians would object. Who wants to pay fifteen to twenty-five dollars for a medical journal of twelve—or fifty-two—issues annually? Of course a ticket to a popular Broadway musical might cost as much, but there is a difference between fun and education even when one is brief and the other enduring.

6. I conclude reluctantly that my five suggestions are almost wholly impractical. This brings me to a sixth suggestion which is perhaps more palatable because it leaves decision to someone else: namely, a national testing laboratory. Inasmuch as this meeting is under the auspices of the Minnesota Heart Association, an affiliate of the American Heart Association, I will restrict my remarks to the problem of evaluation of drugs alleged to be of value in the treatment of patients with cardiovascular diseases. This situation represents only a small part of the whole; but if such a laboratory were established and maintained by the American Heart Association, its orderly investiga-

tions might persuade appropriate organizations to follow a similar program. I refer to representative bodies in the fields of nutrition, psychiatry, neurology, and gastroenterology, for example.

Such a program of study would be well within the stated objectives of the American Heart Association. It would require indefinite tenure for investigators to protect them against the actions which might arise from disdain and hostility. It would require good scientific minds dedicated to the principle that a negative result is as good as a positive one. The results achieved would certainly protect the public in general and physicians specifically against poor investments in drugs. I suspect it would find little attractiveness for those qualified for such study, but perhaps there is enough missionary zeal in the United States to make possible the implementation of such a program. An agency other than the American Heart Association is currently establishing such a program.

Now I abandon an introduction which has threatened to swallow the entire presentation and return to the specific subject which has been assigned to me.

Atherosclerosis

The most important peripheral vascular disease is atherosclerosis. The idea that man is a composite of that which he eats is certainly not new. Actually, the idea is an obvious expression of ignorance. However, the proposition that atherosclerosis is a manifestation of the fat which man eats must be considered. Almost all of you know that this idea is based on the observation that atheromatosis can be produced in some animals by feeding them excessive amounts of cholesterol and fats. It has seemed a relatively easy transition from experiments on animals to conclusions relative to man.

Too little consideration has been given to the fact that patients eating essentially the same diet become ill and die in diverse ways and periods of life. One may question why my mother died of acute myocardial infarction at the age of forty-eight and why my father has no clinical evidence of atherosclerosis at the age of ninety-two. A study of ancestors and siblings in this instance reveals clearly a genetic factor of great importance. Also, no one has explained satisfactorily to me why atherosclerosis involves the coronary and cerebral arteries and those of the legs, while it leaves unaffected the pulmonary arteries and those in the upper extremities.

But even within the scope of the experiments so widely reverenced, a fundamental omission exists. In applying the findings in animals to the problem in man there has been, for the most part, neglect of the observation that arterial occlusion, which may be a grave complication of atherosclerosis, almost never occurs in the animals in which atheromatosis has been induced. This provokes an important question as to whether or not benign atheromatosis of animals is the equivalent, in an etiologic way, to complicated atheromatosis of man. I do not know the answer to this question, but as of this date I see no significant evidence that it is "yes." I cannot recommend a low-fat diet for everyone in September, 1957.

I can recommend that attempts be made to reduce the concentration of fats in the blood when they are persistently and significantly increased in amount, although even in this situation there is more presumption of a causal association than evidence of it. Hardly anyone likes to restrict or modify his diet; hence I suggest beginning with the ingestion of nicotinic acid in the amount of 500 mg. six times daily. The anticipated flush is seldom troublesome after the first few doses of nicotinic acid, and many times the concentration of fats in the blood is reduced. If the administration of nicotinic acid fails to reduce the fat in the blood in four weeks, it may be replaced by administration of one of the plant sterols, such as sitosterol. If this is ineffective in a period of several weeks, it too may be abandoned.

We arrive now at consideration of the use of unsaturated fats such as safflower oil or corn oil, which needs more study. I anticipate a flood of literature on the use of unsaturated fats; indeed, the flood seems already here. It is clear that there have been and will be exaggerated claims. I have before me an advertisement for linodoxine emulsion which reads as follows: "Useful prophylactically or therapeutically in patients who either show elevation of serum cholesterol or fall into one or more of the following clinical categories: male patients with precordial pain; overweight middle-aged patients of both sexes; patients with visibly tortuous superficial arteries; patients with elevated blood pressure." This statement is for the most part pure nonsense. It is calculated to assure the widest possible use of linodoxine emulsion regardless of need. I certainly will abstain from the use of it. The advertisement should insult the intelligence of every physician, but it has much in common with material which comes to you repeatedly.

Nevertheless, I do believe the trial use of unsaturated fats has a place in an attempt to reduce the concentration of cholesterol in the blood when the increased value persists despite the countermeasures mentioned previously.

A final measure which may be effective in reducing the amount of fat in the blood is a diet restricted in fat to about 30 grams. Among patients, the respect for such a diet varies widely —from complete adherence to complete negligence. This is a situation commonly noted in attempts at reduction of weight by means of diet. I have a simple philosophy in this regard: the major function of a physician is to recommend; it does not include police responsibility.

Hypertension

The next most important peripheral vascular disease is hypertension. When this is a significant problem, original studies should include the search for pheochromocytoma, aldosteronism, coarctation of the aorta, and unilateral renal disease. The treatment of essential hypertension is very satisfactory, although active preparations used to correct hypertension have other effects besides reduction of blood pressure. I have observed lupus erythematosus and rheumatoid arthritis from hydralazine (apresoline), mental depression from rauwolfia, and psychosis from mecamylamine (inversine). These and other reactions, however, are far from sufficient to justify avoidance of the use of these drugs. Incidentally, I believe that apresoline, the various rauwolfia preparations, and the ganglion-blocking agents are the only drugs which are satisfactory in the treatment of chronic essential hypertension.

But for the past five years, my chief concern regarding hypertension has been the failure of physicians to appreciate the need for treatment. Too often they delay action until distressing complications have developed, or they permit patients to abandon a regimen of treatment which has been established. There is common agreement that patients who have hypertension with retinopathy or with papilledema need treatment, and studies show sharply reduced mortality as a result of adequate measures. It is about the need for treatment of patients with uncomplicated hypertension that disagreement lingers. My own opinion may be simply stated: Hypertension is an abnormal condition which is a notorious provoker of atheromatosis; it should be treated. On a number of occasions when I have participated in panels dealing with hypertension, some physician has disagreed with my opinion. I have always asked him whether he would prefer to have hypertension or normal blood pressure; he has always replied that he would prefer to have normal blood pressure if he had no significant side reactions from drugs. I have never been quite certain of the reason for a physician's desiring to have normal blood pressure when he did not believe his patient needed to have it.

I will turn from these brief remarks upon essential hypertension with the statement that of course there should be continuing study to determine the cause of this disease.

Arterial Occlusion and Aneurysm

The third most common peripheral vascular disease is comprised of complications of atherosclerosis, namely arterial occlusion and aneurysm. The prognosis for untreated aneurysm is so grim that operation is logically advisable. I bow with respect to my surgical colleagues who have accomplished so much in this field. For the most part, aneurysm is a disease of the aged who have atherosclerosis in other areas. This situation and the inherent risk of operation which is present, without reference to age, promote a surgical mortality which is substantial. However, the risk of operation is less than the risk of withholding surgical treatment in most instances. Some patients who believe their lives have almost reached a full cycle prefer the risk of going without surgical treatment; their opinions are to be respected if they are based on complete information.

You will hear much during this program relative to re-establishment of circulation in cases of segmental occlusion of the abdominal aorta or the iliac or femoral arteries. Here, again, I observe a remarkable example of the great skill of surgeons and I am gladdened at the immediate results, which are extremely good in most instances. All of us who participate in this field of medicine are aware that a surgeon cannot cure atherosclerosis. He may, however, correct an impediment to the flow of blood, an accomplishment which is gratifying to patient and physician alike. We have noted on occasion that, at a variable period following operation, such patients may have angina pectoris, acute myocardial infarction, or cerebral arterial insufficiency, which may be lethal or of grave importance. In considering operations for occlusion of the abdominal aorta, the physician must measure the operative and postoperative risk against the degree of current disability; and the patient should share in this evaluation.

Anticoagulants, such as heparin and dicumarol and drugs having similar action, have achieved an established place in the treatment of vascular disease. Part of this situation is owing to sound observation and part to sound theoretical considerations. In substantial degree, we lack satisfying evidence of their value, based on case-matching. The difficulty in obtaining this is great, because of the infrequency of finding two patients who have exactly similar diseases.

I am concerned particularly about the use of dicumarol and drugs of similar action. Our first report on the use of dicumarol was published in 1942, and during the fifteen years which have elapsed, a multitude of reports have been published and presented orally. Still I must regretfully say that many physicians remain uninformed about, and many laboratories remain unable to report accurately on, prothrombin activity. Consequently the prothrombin activity in blood is reduced too much or not enough. It is distressing to note that many patients take dicumarol without adequate laboratory control by determination of prothrombin activity.

Sympathectomy

Some comment should be made on the use of sympathectomy for peripheral vascular disease. Appropriate sympathectomy almost invariably relieves Raynaud's disease of the feet. For reasons wholly unknown to me it fails in at least a third of the cases in which it is performed for Raynaud's disease of the hands. When Raynaud's syndrome is associated with acrosclerosis, sympathectomy is valueless.

As a method of treating essential hypertension, sympathectomy has been almost wholly abandoned. This is because it produces satisfactory results in not more than a third of the instances in which it is used against essential hypertension, and because treatment of this disease with drugs has improved enormously. There are, I believe, only two indications for the use of sympathectomy in the treatment of hypertension: failure of adequate control with drugs, and disinclination on the part of a patient to undertake a program of treatment with drugs which must be carried out indefinitely.

For patients with impaired circulation to the legs, sympathectomy is used somewhat less frequently than it used to be, chiefly because surgical measures are applied directly to the arteries more frequently. In most instances we do not know what the clinical course of arteriosclerosis obliterans will be. Often it is a very benign disease; occasionally it necessitates amputation. People with arteriosclerosis obliterans die from the complications of atheromatosis of the cerebral and coronary arteries more often than from the primary affliction itself. I believe it is true that the major aim of all treatment including sympathectomy is preservation of the extremities until the patient dies of some other condition. Sympathectomy for impaired circulation to the legs is on a sound theoretical basis, for it improves circulation to the skin, in which gangrene ordinarily begins if it occurs. Unfortunately for the desire for complete information, there has been no case-matching to indicate whether or not sympathectomy saves limbs; but I believe it does in many instances.

Sympathectomy is not indicated for the relief of intermittent claudication, which seldom is lessened thereby.

Miscellaneous Conditions

The symptom of claudication is annoying; but owing to the lack of need to walk far, it seldom is accounted disabling. In consideration of this symptom I must say with candor that there is little evidence that any medication influences claudication favorably.

The treatment of sudden arterial occlusion—particularly that resulting from emboli—has been signally improved, although the evidence is not entirely clear that either surgical or nonsurgical treatment has the greater virtue in many instances. The treatment of varices by excision is a forward step of much importance. The use of cortisone or similar steroids has improved remarkably the outlook for patients with polyarteritis and cranial arteritis.

Comment

As I consider again what I have written, I find a substantial amount of pessimism which I had not intended, nor do I intend to close this

(Continued on Page 265)

The Physiologic Action of Ganglionic and Adrenergic Blocking Agents on the Peripheral Circulation

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PERIPHERAL VASCULAR DISEASE affecting the extremities can be divided into two major components: vasospastic and obliterative. Vasospastic vascular disease may be primary or may be associated with obliterative disease inducing symptoms referable only to the skin. It is uncertain, at the present time, whether the vasospasm is due to the hypersensitivity of the terminal arteries, arterioles and precapillary sphincters in association with a normal intensity of sympathetic vasoconstrictor impulses and/or circulating constrictor substances; or whether it is due to a heightened intensity of these impulses or substances. In either case, a basic mode of attack on the vasospastic component is blockade either of the neurogenic pathways or of the receptor areas on the smooth muscle in the walls of the arterioles.

The obliterative component may be due to thickening of the intima often with segmental thrombosis superimposed on an area of intimal damage. The symptoms may be referred to the skin or muscle. At the moment there seems to be no appropriate pharmacologic attack on the obliterative process itself although other papers in this symposium discuss prophylaxis of the atherosclerotic type of obliterative vascular disease. However, it is probable that the development of collateral circulation around any region of segmental occlusion may be hastened by inducing a prolonged demand for blood distal to this area. By this process some long term improvement of even the obliterative vascular disease may be brought about. This can be accomplished in part by maintaining a vasodilation in the terminal arteries and arterioles distal to the occlusion by the use of blocking and vasodilator agents, alone or in combination.

Three categories of drugs are being used at present in inducing vasodilation in patients with vascular disease. These include: (a) the adrenergic blocking drugs, (b) the ganglionic blocking drugs and (c) drugs having a direct vasodilator action on the arterioles. The following is a discussion of the physiologic effects and some of the pharmacologic properties of these agents.

Adrenergic Blocking Drugs

Intra-arterial or intravenous injections of the adrenergic blocking drugs may induce only a brief vasodilation; yet, by observing the effect of the blocking agents on the vasoconstrictor responses induced by adrenergic stimulation, their pharmacologic action can be demonstrated to persist for several hours following their injection.[1,2]

Adrenergic stimulation and blockade in cutaneous vascular beds.—Intra-arterial injections of 0.1 to 1.0 μgm. of arterenol (norepinephrine) or of epinephrine (methyl arterenol) cause vasoconstriction which may reduce the blood flow almost to zero. After two to three minutes, the blood flow gradually returns to the control level without overshoot. Stimulation of the lumbar sympathetic chain induces a similar response. Intra-arterial administration of small doses of one of the adrenergic blocking drugs diminishes the constrictor response to the above forms of adrenergic stimulation. The constrictor response is progressively diminished as the dose of the adrenergic blocking drug is increased. When 3.0 mg/kg of Ilidar® or 0.3 mg/kg of Dibenzyline® is administered, this response is abolished. There is no evidence of any significant reversal, that is, conversion of the constrictor response to a dilator response.[3]

Presented at the Symposium on Peripheral Vascular Disease co-sponsored by the Minnesota Heart Association and the Mayo Foundation, Rochester, Minnesota, September 24, 1957.

From the Departments of Physiology and Pharmacology, and Internal Medicine, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina.

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Adrenergic stimulation in skeletal muscle beds.—In man as well as in the dog, intra-arterial injections of minute doses of epinephrine (0.01-0.1 μgm.) cause a slight evanescent vasodilation. Injections of doses as large as 1.0 μgm. may cause a dilation in about 10 per cent of the subjects but in the remainder, a fairly prominent vasoconstriction occurs. In those animals in which 1.0 μgm. induces vasodilation, successive 1.0 μgm. injections convert this to a prominent vasoconstrictor response. Larger doses, that is, 3.0 to 10 μgm., of epinephrine intra-arterially consistently cause a marked vasoconstriction in the skeletal muscle beds of both the dog and man.[4-10] The magnitude of the vasoconstrictor response following intra-arterial injections of all sized doses of arterenol increases linearly with the logarithm of the dose. The constrictor response to epinephrine and often that to the larger doses of arterenol is followed frequently by a phase of vasodilation.[10]

In the dog, stimulation of the lumbar sympathetic chain may produce a fleeting initial dilation but this is usually followed by a prominent constrictor response. Upon cessation of the stimulation a brief phase of dilation may ensue. Administration of atropine abolishes both the initial and terminal dilator responses and after this drug, lumbar sympathetic stimulation produces only vasoconstriction.[10]

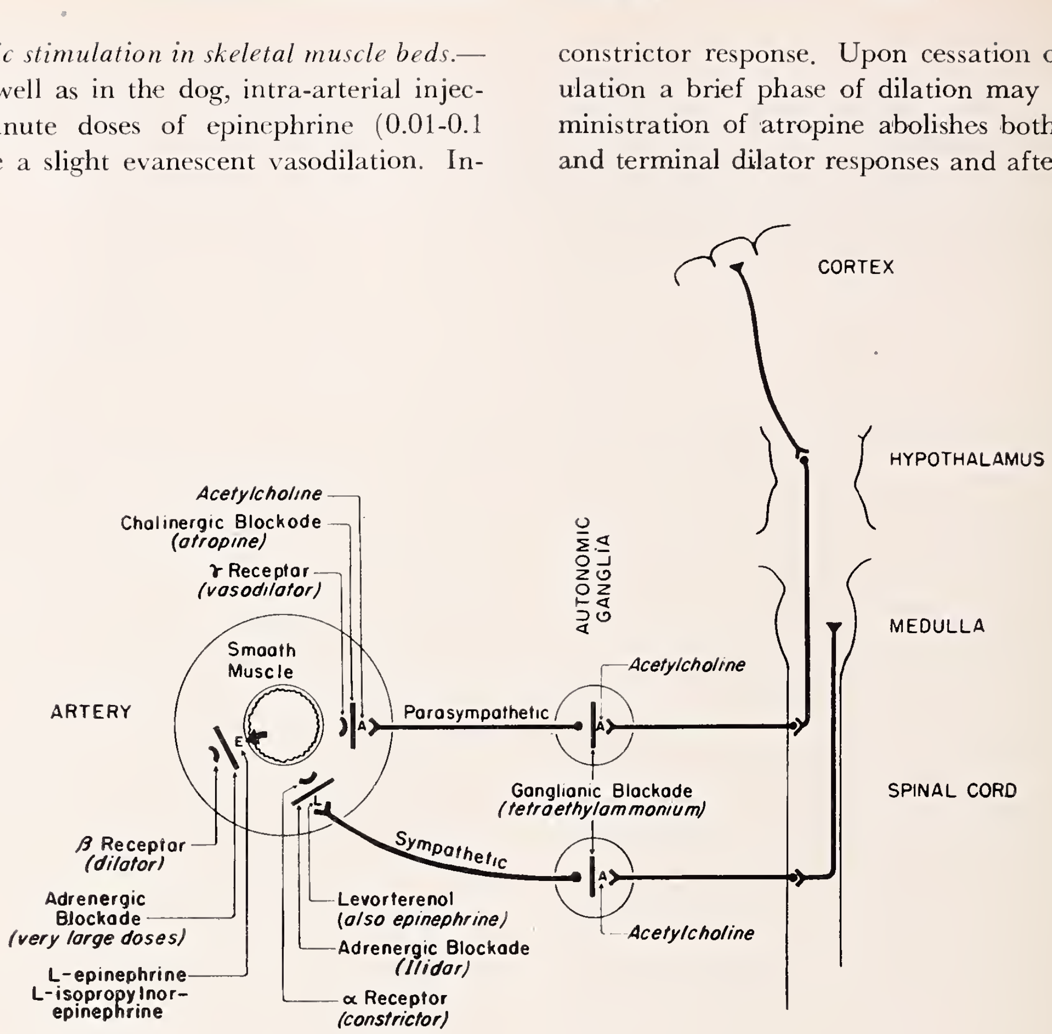
CORTEX
HYPOTHALAMUS
MEDULLA
SPINAL CORD
AUTONOMIC GANGLIA
Acetylcholine
Cholinergic Blockade (atropine)
γ Receptor (vasodilator)
Smooth Muscle
ARTERY
E
A
L
Parasympathetic
Sympathetic
Acetylcholine
Ganglionic Blockade (tetraethylammonium)
Acetylcholine
β Receptor (dilator)
Adrenergic Blockade (very large doses)
L-epinephrine
L-isopropylnor-epinephrine
Levorterenol (also epinephrine)
Adrenergic Blockade (Ilidar)
α Receptor (constrictor)

Fig. 1. Schematic diagram of the various receptors in the smooth muscle of the wall of a blood vessel (α, β, γ), of the autonomic ganglionic synapses, and of the sites at which adrenergic and ganglionic blocking drugs act.

Adrenergic blockade in skeletal muscle beds.—Small intra-arterial doses of either of the adrenergic blocking drugs, Ilidar® (0.3 mg/kg) and Dibenzyline® (0.1 mg/kg), diminish the constrictor response to arterenol and that to lumbar sympathetic nerve stimulation, and convert the epinephrine constrictor response to vasodilation. Larger intra-arterial doses of the adrenergic blocking drugs, Ilidar® (30 mg/kg) and Dibenzyline® (3 to 10 mg/kg), abolish the constrictor responses to lumbar sympathetic stimulation and to arterenol. These doses may unmask a slight arterenol dilator response, but only serve to intensify the vasodilator responses to epinephine. Administration of atropine at this time has no effect on the dilator response to epinephrine but abolishes any vasodilator response to lumbar sympathetic nerve stimulation or to arterenol that may have been unmasked by the adrenergic blocking drug.[10]

Extremely large doses of the adrenergic blocking drugs appear at times to abolish the dilator response to epinephrine.[10] This is a true blockade in the case of Ilidar. However, with dibenzyline this is only apparent and is due to the development of maximal vasodilation which appears to persist for an extremely long time following the injection of epinephrine during the existence of a high degree of adrenergic blockade. Restoration of some degree of vasomotor tone by the intra-arterial injection of pitressin restores the vasodilator capacity as shown by the fact that epinephrine injected following pitressin again induces a vasodilator response.[5]

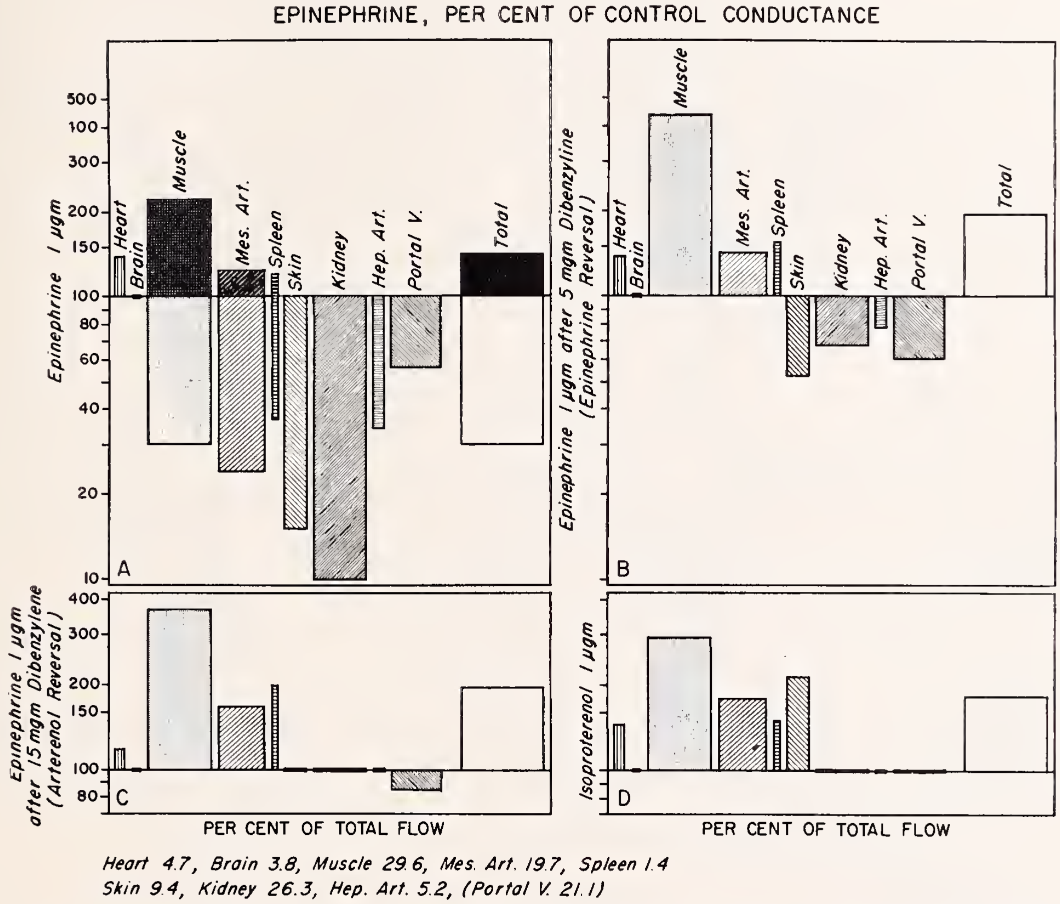

Fig. 2. Response in various vascular beds of the dog to intra-arterial injections of 1 µg. of epinephrine: (A) during a control state, (B) after 5 mg. of Dibenzyline® (or 15 mg. of Ilidar®) intra-arterially, (C) after 15 mg. of Dibenzyline® (or 50 to 100 mg. of Ilidar) intra-arterially, (D) responses to 1 µg. of Isoproterenol (Isopropylnorepinephrine, Isuprel®) intra-arterially during the control state. Ordinate scale indicates response expressed as per cent of control conductance. Conductance = Flow in ml./pressure in mm. Hg.; 100 = no response, greater than 100 = vasodilation, less than 100 = vasoconstrcition. Abscissal scale is drawn approximately proportional to the per cent of the cardiac output flowing in each vascular bed. Total = the calculated change in total body conductance, computed from the sum of the conductance change in each bed, weighted by the per cent of the cardiac output flowing in that bed. Black on white symbols, principally below the 100 line in A, = first or primary response; white on black = secondary response.

Vascular receptors.—From the above studies it can be concluded that in cutaneous vascular beds there are predominantly alpha (constrictor) adrenergic receptors which are stimulated approximately equally by epinephrine, arterenol and through the lumbar sympathetic fibers. These receptors are moderately readily blocked by the adrenergic blocking drugs. In the skeletal muscle vascular bed, alpha adrenergic receptors are also present and are innervated by the lumbar sympathetic fibers. Also present in the vasculature of skeletal muscle are beta receptors which respond to epinephrine and which can also be shown to respond to isoproterenol (isopropylarterenol). These receptors are not innervated and are highly resistant to blockade by the adrenergic blocking drugs. Such receptors are only minimally present in the vasculature of the skin. In the muscle vascular beds it is obvious that arterenol or an arterenol-like substance rather than epinephrine is the mediator of the sympathetic constrictor impulses since there is no significant reversal of either the arterenol-induced or sympathetic nerve-induced constriction. Also present in skeletal muscle are gamma, or cholinergic, receptors which respond to intra-arterial injections of acetylcholine and which are innervated by autonomic fibers traveling in the lumbar sympathetic chain. These receptors are not affected appreciably by the adrenergic blocking drugs but are readily blocked by atropine.

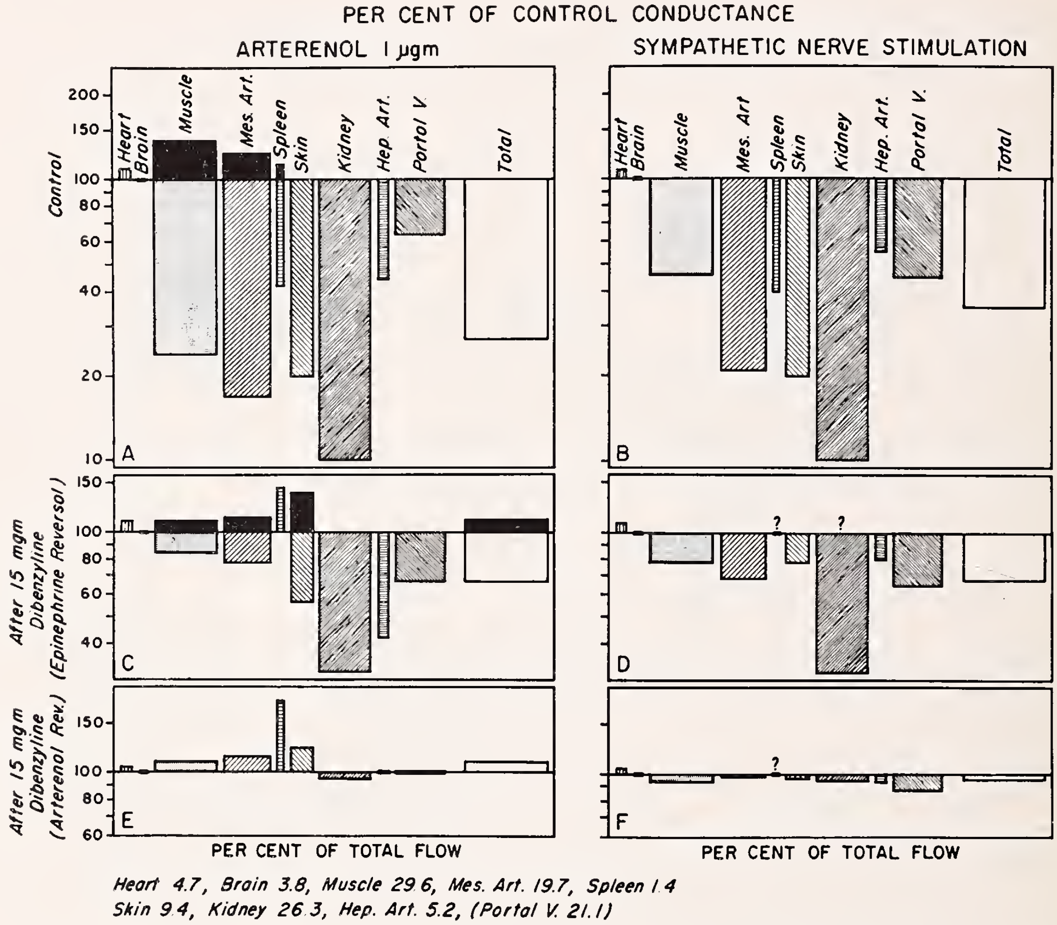

Fig. 3. Responses in various vascular beds of the dog to intra-arterial injections of 1 μg. of l-arterenol (l-norepinephrine), A, C and E, and to lumbar sympathetic nerve stimulation, B, D and F. A and B in a control state, C and D after 5 mg. of Dibenzyline® intra-arterially and E and F after 15 mg. of Dibenzyline® intra-arterially. Black on white symbols, principally below the 100 per cent line, indicate primary response, and white on black, all of which are above the 100 per cent line, indicate secondary response.

Extensive studies by Folkow and his collaborators[11] demonstrate that the sympathetically innervated alpha (adrenergic) receptors respond to the medullary homeostatic mechanisms in the regulation of arterial pressure, whereas the sympathetically innervated gamma (cholinergic) receptors do not. On the other hand, the gamma receptors are under the control of the hypothalamus and cortex and probably serve to regulate the blood flow in skeletal muscle in accordance with or in anticipation of skeletal muscle activity. Adjustment of the blood flow to the needs of skeletal muscle does not appear to be mediated by way of the alpha (adrenergic) receptors or by way of the adrenergic mediated autonomic fibers in the lum-

(Turn to Page 245)

bar chain. The above receptors are illustrated in Figure 1 which also shows schematically the site of action of the adrenergic blocking substances on the alpha receptors and the site of action of the cholinergic blocking agents on the gamma receptors.

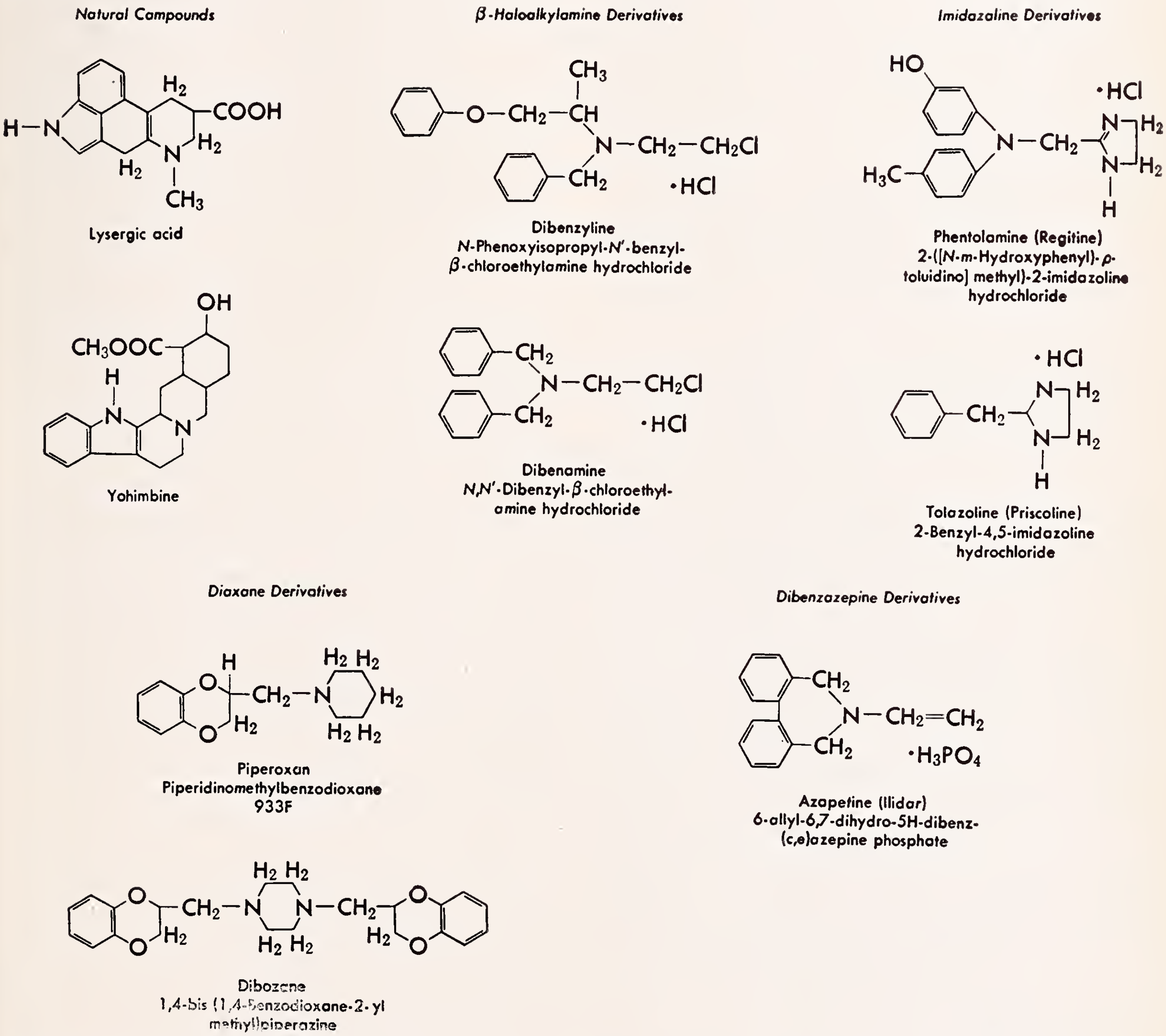
Natural Compounds
Lysergic acid
Yohimbine
β-Haloalkylamine Derivatives
Dibenzyline
N-Phenoxyisopropyl-N′-benzyl-β-chloroethylamine hydrochloride
Dibenamine
N,N′-Dibenzyl-β-chloroethylamine hydrochloride
Imidazoline Derivatives
Phentolamine (Regitine)
2-([N-m-Hydroxyphenyl)-p-toluidino] methyl)-2-imidazoline hydrochloride
Tolazoline (Priscoline)
2-Benzyl-4,5-imidazoline hydrochloride
Dioxane Derivatives
Piperoxan
Piperidinomethylbenzodioxane
933F
Dibozane
1,4-bis (1,4-benzodioxane-2-yl methyl)piperazine
Dibenzazepine Derivatives
Azapetine (Ilidar)
6-allyl-6,7-dihydro-5H-dibenz-(c,e)azepine phosphate

Fig. 4. Structural formulae for various substances which possess adrenergic blocking action (Reproduced from Drill, Pharmacology in Medicine[17]).

Effects of Adrenergic Blockade and Adrenergic Stimulation on Other Vascular Beds.—In order to appreciate the potential usefulness of the adrenergic and ganglionic blocking drugs, it is desirable to know what the effects of adrenergic stimulation and blockade would be in the other major vascular beds in the body. Epinephrine causes primary vasoconstriction in skeletal muscle, in the splenic, cutaneous, and renal vascular beds, in the mesenteric and hepatic arteries and in the hepatic portal venous vascular beds (Fig. 2A). Secondary dilation may occur in the skeletal muscle and mesenteric arterial beds. No response occurs in the cerebral bed; and in the coronary bed a mild vasodilation may be elicited. During adrenergic blockade, the constrictor responses are progressively reduced and, in the case of skeletal muscle, mesenteric artery and splenic beds, the constrictor responses are reversed, that is, converted to vasodilator responses (Fig. 2B). Because of the overall magnitude of the flow in these beds as compared with that in the other beds, the net effect on the total vascular resistance in the body after appropriate doses of the adrenergic blocking drugs given intravenously is vasodilation. Arterenol and sympathetic nerve stimulation cause vasoconstriction in the various vascular beds in a manner exactly comparable to that of epinephrine (Fig. 3). Reversal of the response to arterenol by Dibenzyline occurs only to a small extent in skeletal muscle, the mesenteric arterial bed, and in the vascular beds of the skin and spleen. Reversal of the adrenergic constrictor response to sympathetic nerve stimulation does not occur.

In view of the above it would appear that moderate doses of the adrenergic blocking drugs might be expected to reduce excessive vasospasm where it exists without causing excessive fall of arterial pressure unless there is an abnormal quantity of epinephrine circulating in the body. If such is present, however, the marked vasodilator responses which would occur in the two large vascular beds, skeletal muscle and the mesenteric bed, might be expected to decrease the total peripheral resistance producing a significant fall in the arterial pressure impairing the cerebral, coronary and renal circulations, in which reversal does not occur.

Available Preparations.—The adrenergic blocking drugs which are available at the present time are indicated in Figure 4. For oral administration, the following preparations are available: azapetine (Ilidar®) 25 mg. tablets, phenoxybenzamine (Dibenzyline®) 10 mg. capsules, and tolazoline (Priscoline®) 25 mg. tablets. A common factor in all of these is a tertiary nitrogen, usually linked directly or through a single carbon atom with one or two benzine rings. The nitrogen may be linked also with an imidazoline ring or with a two-carbon aliphatic chain containing chlorine such as is seen in Dibenzyline,® or an unsaturated linkage such as is seen in Ilidar.

Evaluation of the Effects of These Drugs in Man.—In man, the blocking effects of these drugs have been studied by recording the cutaneous blood flow determined with the thermocouple, measuring the skin temperature on the tips of the toes and fingers while the subjects are exposed in the constant temperature room in which the air is circulating relatively rapidly (25-50 feet/min. at an ambient temperature of 20°C). This technique is the equivalent of the calorimetric method of measuring blood flow and the rate of circulation can be approximated closely by determining the temperature of the skin when it comes into equilibrium. When the skin temperature approximates the room temperature, the blood flow is extremely low. As the skin temperature approximates body temperature, the blood flow must increase as much as 100-fold over that when the skin temperature is at its minimal point. A minimum rate of blood flow, that is, maximal vasoconstriction, may be induced by exposing subjects to this environmental temperature while they are clad only in shorts or a slip for from a half to one and one-half hours, provided they have not eaten or exercised during the hour or two prior to the exposure.

The ability of the adrenergic blocking drugs to block this neurogenically induced vasoconstriction can be studied by slowly administering the drugs intravenously over the course of a half hour while maintaining the exposure to the cool environment. Figure 5 shows the results of such studies on ten medical students. The doses of the adrenergic blocking drugs were the maximal doses which the student could tolerate without untoward symptoms during the study and without the occurrence of excessive postural hypotension following the study. On the basis of this choice of doses, Ilidar® was the most effective; Priscoline® and Regitine® less effective, respectively; and Dibenzyline® almost ineffective except in the upper extremities. Consistently in studies such as this, the vasodilator response has been more marked in the upper than in the lower extremities.[12,13]

Clinical Use of the Drugs.—The adrenergic blocking drugs may be given intra-arterially in acute emergencies though it is perhaps safer to administer them by intravenous drip. For prolonged administration it is more satisfactory to give the drugs orally. In oral administration one tablet four times a day is prescribed initially and every two to three days the dose is increased by one tablet until the maximum tolerated dose is reached. In the case of Ilidar this is usually three to four tablets four times a day.

The principal side effects which may be noted with these drugs are those due to postural hypotension which may ensue when the maximum dosage is used. The symptoms include dizziness, nausea and fainting. Such hypotension is rarely severe, but should be avoided in patients with atherosclerosis. This difficulty has been more prominent with Dibenzyline® than with the other drugs due, perhaps, in part to the more prolonged action of this substance and its greater tendency to cause epinephrine reversal. Tachycardia and palpitation have been reported by many investigators but when the patient gradually adjusts the dose, palpitation has not been a prominent symptom. In case a patient develops an intercurrent infection, particularly a viral infection, it is well to reduce the dose or discontinue the use of these drugs until full recovery from the infection has occurred, since the infection tends to potentiate the postural hypotension induced by the drugs. Cutis anserina, that is, gooseflesh, formication and chilliness are prominent with Priscoline® but do not occur with the other drugs. All these drugs may occasionally produce gastrointestinal disturbances. About 2 per cent of the patients taking Ilidar® will experience a drug fever, characterized by flu-like symptoms of aching, chills and fever amounting to 101-103°F. These symptoms subside within eight to twenty-four hours after discontinuing the drug. This drug fever does not usually occur until after two or more weeks of continuous administration. Should such symptoms develop, the patient should be shifted to another drug since the sensitivity appears to persist indefinitely.[14]

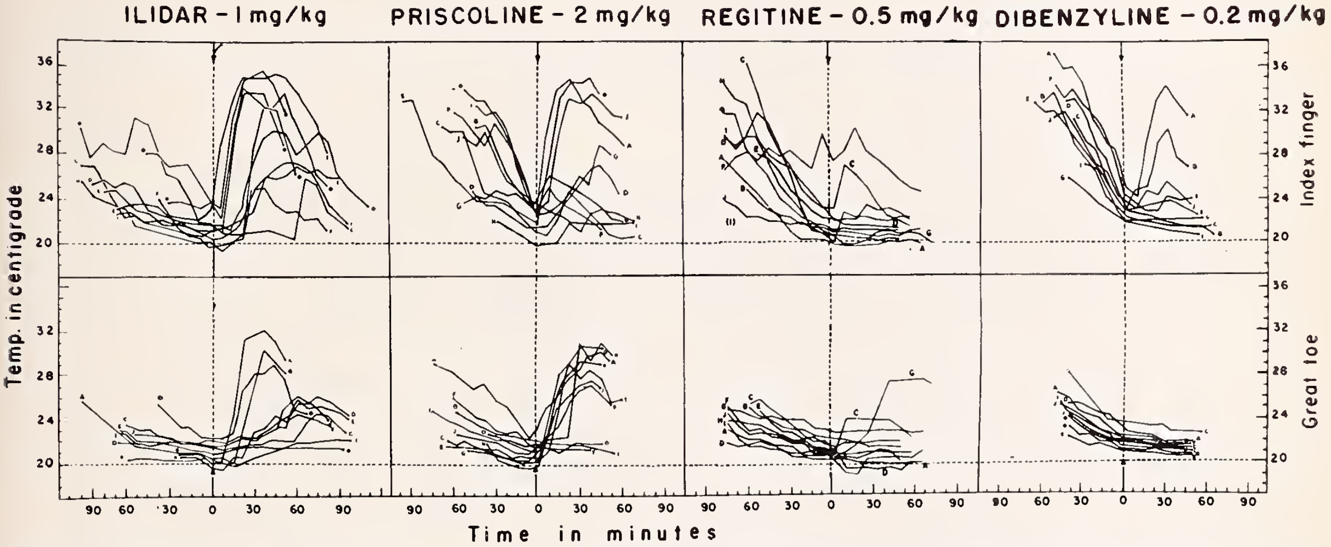

Fig. 5. Responses to intravenous infusions of four adrenergic blocking drugs in each of ten normal medical students. Ordinate scale indicates skin temperature of an index finger (upper curves); and the large toe (lower curves) expressed in degress Centigrade. Room temperature 20°C., range ± 0.3°C., air velocity 50 feet per second. Throughout the study the subjects were fasted, and were exposed, clad only in shorts. Temperatures close to room temperature indicate maximal vasoconstriction; temperatures close to 36°C., maximal vasodilation. (Reproduced in modified form from references 12 and 13).

In our hands, improvement of vasospastic symptoms and, with prolonged use, some improvement of the walking distance due to development of collateral circulation, has been obtained in a large proportion of patients.[14,15] A small number of patients in whom the vasospasm is so severe that it cannot be overcome without the occurrence of severe side effects may require sympathectomy. We have not seen any immediate relief of the symptom of intermittent claudication with the use of any of these drugs. This is of interest since, because of the phenomenon of epinephrine reversal, one might anticipate a more marked vasodilator effect in skeletal muscle than in skin. However, it is probable that the vasodilator response to ischemia in skeletal muscle produces a maximal vasodilator effect which cannot be enhanced by blockade of the sympathetic fibers or of circulating adrenergic substances.

Ganglionic Blockade

Unlike the adrenergic blocking drugs, intra-arterial injections of the ganglionic blocking drugs have no vasodilator effect on the terminal arteries, arterioles or precapillary sphincters. In fact, a slight vasoconstriction may be induced by the intra-arterial injections. These substances do not have any effect on the responses to the intra-arterial injections of the adrenergic substances, nor on the responses to postganglionic stimulation of the sympathetic fibers. The ganglionic blocking drugs do, however, block transmission at the ganglion of both the sympathetic (adrenergic) and the parasympathetic (cholinergic) autonomic impulses. Presumably these ganglionic blocking agents also block the release of adrenalin from the adrenal gland in response to splanchnic nerve stimulation. The site of action of the ganglionic blocking drugs is portrayed in Figures 1 and 6.

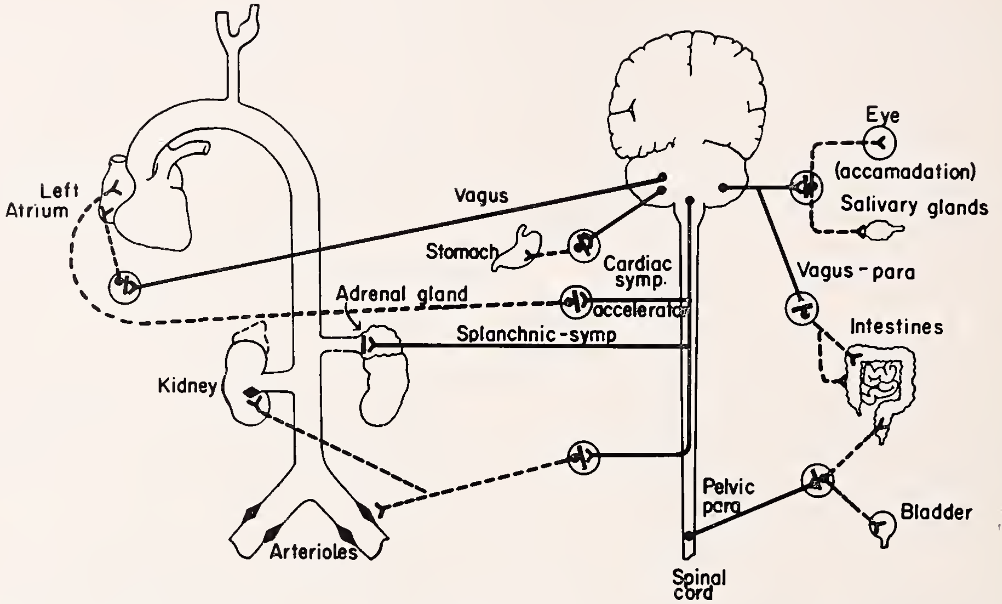

Fig. 6. Ganglionic blockade. Diagram of the sites of action of the ganglionic blocking drugs on the sympathetic (left side exclusive of the vagus) and parasympathetic (right side) divisions of the autonomic nervous system. Dashed lines indicate the fibers in which the number of impulses is reduced as a result of the blockade, solid line indicates neuronal activity which is blocked and heavy solid bar indicates blockade. (Reproduced from reference 18). Blockade of the vagus to the heart would lead to speeding of the heart, but this is counterbalanced by blockade of the sympathetic fibers which would lead to cardiac slowing. See text for further discussion.

Available Preparations.—The various preparations which have been used experimentally in the past and which are now available for clinical use are shown in Figure 7. All of these are effective by the parenteral route. By the oral route Ansolysen,® Inversine® and Ecolid® are quite effective, hexamethonium is slightly effective, and the others are without significant effect. For oral administration the following are available: hexamethonium 125 and 250 mg. tablets; pentolinium (Ansolysen®) 20, 40 and 100 mg. tablets; mecaamylamine (Inversine®) 2.5 and 10 mg. tablets and chlorisondamine (Ecolid®) 25 mg. tablets.

Physiologic Studies in Man.—The relative effectiveness of the ganglionic blocking drugs in man can be studied by the same technique as that described above for the adrenergic blocking drugs. The responses to some doses which appeared to be reasonably safe are illustrated in Figure 8. Etamon,® Arfonad® and hexamethonium appear to be the most consistently effective of these particular agents. In contrast to the adrenergic blocking drugs, the ganglionic blocking drugs appear to be more effective on the lower extremities. No satisfactory explanation for the difference in the effectiveness of the adrenergic and ganglionic blocking drugs on the upper and lower extremities exists. Since the ganglionic blocking drugs can exert an effect only on neurogenic tone, it is possible that the difference in the responses to the two groups of blocking drugs may be interpreted as indicating that neurogenic control of vasoconstriction is more prominent in the lower than in the upper extremities. Conversely, it is possible that circulating adrenergic constrictor substances are relatively more important in the upper extremities. If this is the case, it might offer one explanation for the clinical observation that sympathectomy is often more effective in relieving vasospasm in the lower than in the upper extremities.

Side Effects.—The side effects of the ganglionic blocking drugs are those which might be expected from blockade of both the sympathetic and parasympathetic portions of the autonomic nervous system. Those due to sympathetic blockade are primarily postural hypotension, as manifested by weakness, dizziness and fainting. This may be experienced particularly when arising suddenly from a lying or sitting position. Hypotensive reactions may occur more readily with the ganglionic than with the adrenergic blocking drugs. Such reactions should be avoided in atherosclerotic patients since they may induce cerebral or myocardial infarction. Blockade of the parasympathetic system leads to dryness of the mouth, blurring of near vision and obstipation as the principal undesirable effects. The blurring of near vision is more prominent in patients approaching the presbyopic age and may necessitate the use of stronger reading glasses. Obstipation is a significant problem and appears to be alleviated most readily by the use of approximately 15 mg. of Prostigmine in the morning and if necessary the additional use of cascara, 4 to 8 cc. at bedtime. If the patient fails

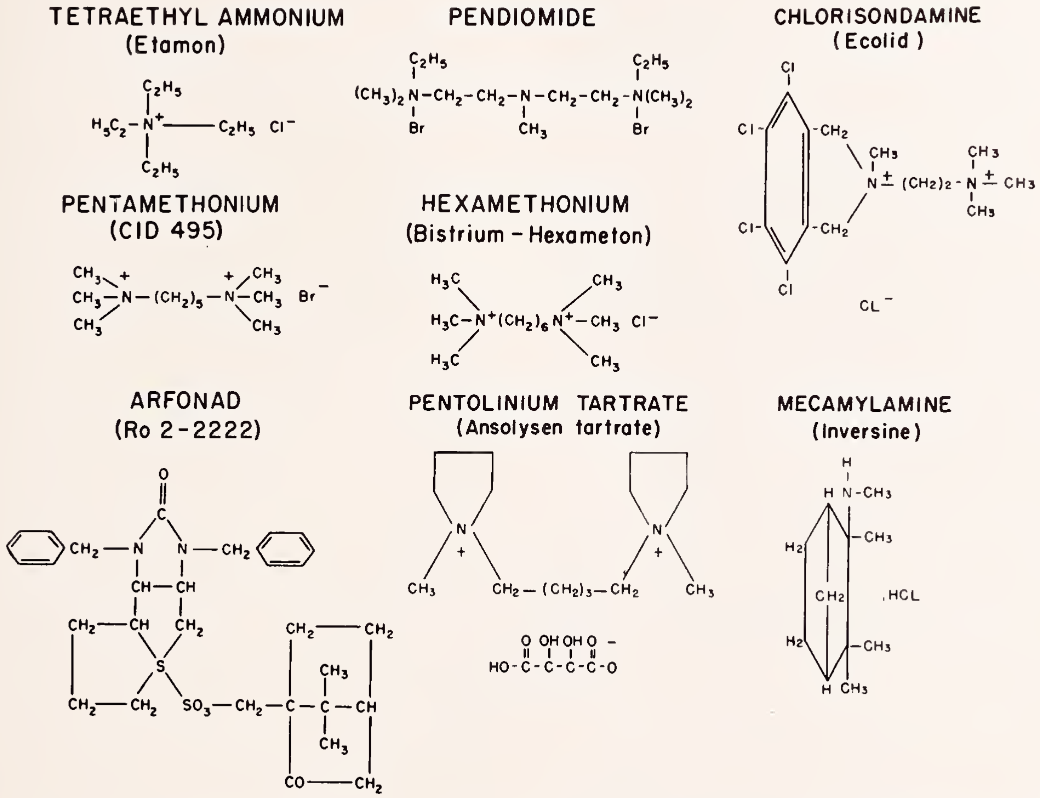
TETRAETHYL AMMONIUM
(Etamon)
PENDIOMIDE
CHLORISONDAMINE
(Ecolid)
PENTAMETHONIUM
(CID 495)
HEXAMETHONIUM
(Bistrium - Hexameton)
ARFONAD
(Ro 2-2222)
PENTOLINIUM TARTRATE
(Ansolysen tartrate)
MECAMYLAMINE
(Inversine)

Fig. 7. Structural formulae for a number of ganglionic blocking drugs which have been used either experimentally or clinically.

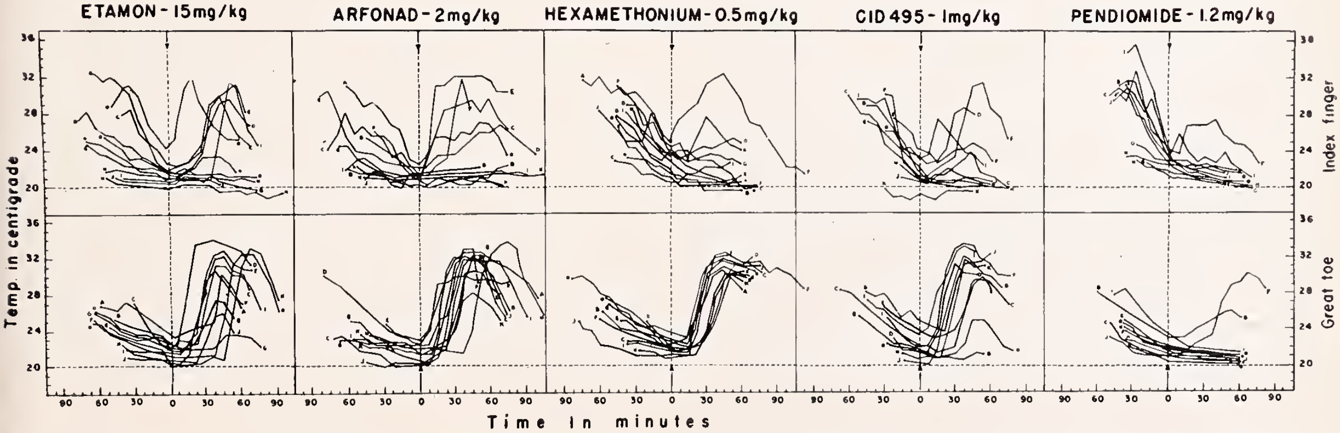

Fig. 8. Responses in ten normal medical students to each of five ganglionic blocking drugs. See legend to Figure 5 for details.

to have a bowel movement, further use of the blocking drug should be suspended until elimination is re-established.[16]

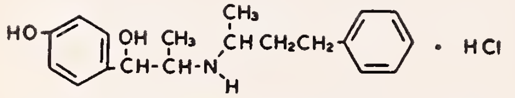

Fig. 9. Structural formula for nylidrin hydrochloride (Arlidin®).

Direct Acting Vasodilators

Nylidin hydrochloride (Arlidin®) is a substance classified in the New and Non-Official Remedies as an adrenergic substance. Part of its structural formula is similar to epinephrine (Fig. 9). This substance is stated to cause vasodilation in muscle but to have no vasodilator effects in cutaneous beds. It was originally promoted for its supposed ability to relieve intermittent claudication by virtue of its muscle vasodilator effects. In our hands, this drug causes vasodilation in the dog in both muscle and cutaneous vascular beds and when given by intravenous drip to human subjects (as in the above studies) causes a prominent vasodilation in the fingers and a weak vasodilation in the toes.

When given to patients by mouth, this substance causes cutaneous vasodilation. In our hands, however, it has no immediate effect on the symptom of intermittent claudication. As with the adrenergic blocking drugs, prolonged use of Arlidin® may stimulate collateral circulation development.

The mechanism of the Arlidin® vasodilation is not established. It is possible that it effects beta receptors in a manner similar to isoproterenol. The effects of adrenergic blockade on this substance are not known. Clinically, it has proven possible to give the drug along with adrenergic blocking drugs with apparently some synergistic effect and without accentuating the hypotensive effects of either drug (work in preparation).

The drug can be administered initially in doses of 6 mg. four times a day and in many patients this can be increased progressively to 18 to 24 mg. four times a day. Postural hypotension does not seem to be a prominent side effect, but patients may complain of considerable nervousness following doses of 12 to 18 mg. When this occurs, the dose should be held at this level or reduced rather than increased. Another similar drug which is undergoing evaluation is Duvadilan.®

Alcohol.—Ethyl alcohol in a dose of 60 to 120 ml. of 100-proof whisky given orally causes a marked cutaneous vasodilator effect in normal human subjects demonstrated by using the above technique of study. This vasodilation is obtained consistently in both upper and lower extremities though it occurs more quickly and completely in the upper extremities. The exact mechanism of the alcohol dilator effect is not known.

Diagnostic and Clinical Usefulness of the Above Drugs

In normal subjects, administration of any of the above drugs in adequate dosage simultaneously with warming of the torso with blankets plus two 30 watt electric heating pads completely relaxes the vasospasm induced in both upper and lower extremities by the exposure to a cool environment. Use of one of these drugs together with body warming therefore provides an effective and convenient means for differentiating vasospasm from obliterative disease affecting the cutaneous circulation in patients. This technique also gives a method for evaluating the severity of obliterative disease, and, by successive studies at weekly to monthly intervals, allows one to evaluate the development of collateral circulation to the skin. Studies of this type are also quite useful in evaluating the effectiveness of sympathectomy. In most cases, postsympathectomy studies yield skin temperatures within 1 to 2° of those predicted during the presympathectomy study (Fig. 10).

Summary and Conclusions

From the above studies it appears that the vasospastic component of peripheral vascular disease may be attacked: (a) by establishing a blockade at the alpha constrictor receptor endings of the terminal arteries, arterioles and precapillary sphincters, by the use of the adrenergic blocking drugs; (b) by relief of the neurogenic component of vasospasm by blocking the sympathetic constrictor impulses at the ganglia by the use of the ganglionic blocking drugs; and (c) by direct induction of vasodilation by the use of substances such as alcohol and Arlidin.® One, or a combination of these drugs may serve effectively in the relief of vasospasm and in stimulating the development of collateral circulation thus providing a medical approach to the control of vascular disease which in many cases is comparable to that obtainable by sympathectomy. Even after sympathectomy some additional benefit may occasionally be obtained by the administration of one of the above drugs.

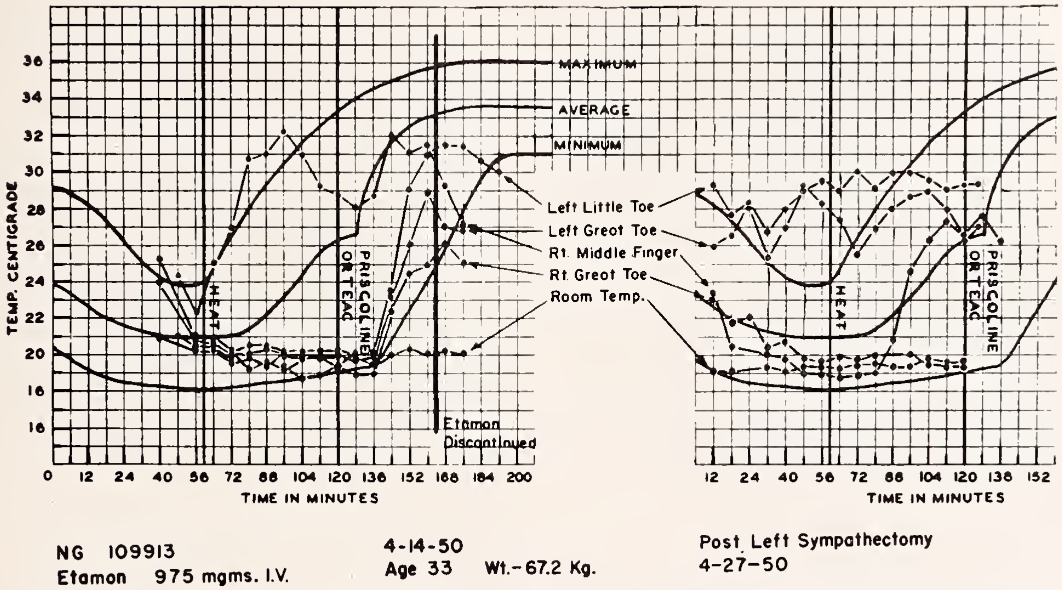

Fig. 10. Skin temperature responses to body warming plus an intravenous infusion of Etamon® in a patient with "vascular disease," prior to sympathectomy—left side. Skin temperatures recorded without the drug in the same patient after a left lumbar sympathectomy. Note that the left toe temperatures stabilized at approximately the same level following the sympathectomy as that obtained in the presympathectomy study.

The principal side effects of these drugs include: (a) postural hypotension, seen with both the adrenergic and the ganglionic blocking drugs; (b) symptoms referrable to the skin, that is, chilliness, cutis anserina and formication, seen with Priscoline;® (c) drug fever seen with Ilidar,® and (d) excessive nervousness seen with Arlidin.®

Appropriate use of these drugs provides: (a) a means for evaluating the degree of vasospasm, and for predicting the probable benefit of drug administration or sympathectomy; (b) with prolonged usage, amelioration of many of the symptoms due to the vasospastic component of peripheral vascular disease; and (c) for development of collateral circulation which may be of assistance in the relief of the obliterative component of the disease.

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(Continued on Page 272)

Fundamentals of Anticoagulant Therapy

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ANTICOAGULANT THERAPY has been employed to prevent intravascular thrombosis for almost twenty years. During the past ten years, its use has been gradually extended on the basis of cumulative experience. Most anticoagulant therapy now is carried out by the employment of one of the coumarin compounds, but in certain situations, heparin is still a valuable anticoagulant and one for which there is currently no available satisfactory substitute.

Heparin

The advantage of heparin is that it produces a therapeutic anticoagulant effect within a few minutes and this effect can be terminated rapidly by stopping administration of the drug, or even more rapidly by the intravenous injection of protamine sulfate. The disadvantages of heparin are that it is costly and it must be administered parenterally. Thus, heparin is not a satisfactory anticoagulant for therapy longer in duration than a few days or weeks. Currently, heparin is used mostly to cover the period between initiation of treatment and achievement of the therapeutic effect of the coumarin compounds, which act more slowly. Administration of both heparin and a coumarin compound is started simultaneously, and the use of heparin is discontinued as soon as tests show that the coumarin effect is established. Short-term anticoagulant therapy occasionally is maintained for a few weeks by use of heparin alone, when for some reason it is inadvisable to employ one of the coumarin compounds.

The degree of anticoagulant effect of fixed doses of heparin varies as greatly among different patients as does that of fixed doses of one of the coumarin compounds. Therefore, regardless of the method by which heparin is administered, it is desirable to check the effect by determination of the coagulation time of whole blood in order to establish the optimal dose and frequency of administration of heparin for each individual patient. However, this is not always practical. When treatment is only given for a few days, fixed dosage schedules for heparin often are employed without determining the degree of its effect.

In my experience, a satisfactory response to heparin for short-term therapy usually is achieved in most patients by the intermittent intravenous injection of 50 mg. every four hours. Less satisfactory effects are obtained by the deep subcutaneous injection of 100 to 200 mg. in a concentrated aqueous solution every twelve hours. This involves considerably fewer injections but introduces the variable of the rate of absorption and increases the variability of effect among different patients, so that more of them will have insufficient anticoagulant effect for certain periods of the day and therefore be unprotected against the condition for which the drug is given. Also, a few will have an excessive effect that may be sufficient to cause serious bleeding. The most satisfactory method of giving heparin in order to achieve the exact degree of anticoagulant effect desired for the individual patient is by the continuous intravenous injection of a dilute solution. The rate of administration is then guided by the coagulation time of whole blood in an effort to maintain the coagulation time between fifteen and twenty-five minutes, or approximately two to four times the normal. This method requires close supervision and much expenditure of time for each patient and is, therefore, not practical in many situations.

Heparin is a powerful anticoagulant drug. The therapeutic effect of adequate doses of heparin is considerably more profound as far as disturbance of the coagulation mechanism is concerned than is that of the coumarin compounds; the danger of hemorrhage is greater with therapeutic doses of heparin than it is with therapeutic doses of the coumarin compounds. When bleeding occurs dur-

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ing the administration of heparin and if it is recognized quickly, it can be terminated more rapidly because the action of heparin can be halted more rapidly, but if the bleeding is internal and therefore not easily recognized, a dangerous situation may develop before the effect of heparin is terminated.

Untoward reactions to heparin other than the possibility of bleeding are few and consist of allergic or anaphylactic phenomena. All such reactions reported to date have occurred among patients who have had allergies to other agents, particularly among those with allergic asthma. Therefore, in any patient who has a history of allergic asthma, cutaneous tests for sensitivity to heparin should be done before large doses of the drug are administered.

Heparin prolongs the prothrombin time as determined by the one-stage technique. When heparin is given to cover the first twenty-four or forty-eight hours of anticoagulant therapy and administration of one of the coumarin compounds is started at the same time, the most satisfactory method of giving the heparin is by intermittent intravenous injections every four hours. A test of the prothrombin time to determine whether the effect of the coumarin compound has reached the therapeutic range should be done only on a sample of blood drawn at least four hours after a dose of heparin has been given, because otherwise the prothrombin time will reflect the combined effect of the coumarin compound and heparin.

Coumarin Compounds

Bishydroxycoumarin (dicumarol) is still the most widely used of the coumarin compounds for anticoagulant effect. Ethyl biscoumacetate (tromexan) has a somewhat more rapid action that terminates somewhat more rapidly when its use is discontinued, but many physicians consider that the effect of tromexan is more erratic, particularly during the first few weeks of administration. When dicumarol is used, the addition of a single dose of 900 to 1,200 mg. of tromexan to the original 300-mg. induction dose of dicumarol will produce a more rapid anticoagulant effect; the prothrombin time will be brought into the therapeutic range within twenty-four hours in about 40 per cent of patients and within forty-eight hours in an additional 50 per cent. With this combination, effects exceeding the therapeutic range will develop within the first few days in approximately 20 per cent of patients.

A recent addition to the coumarin compounds available for therapeutic use is warfarin (coumadin) sodium. Warfarin sodium is soluble in water to a far greater extent than are any of the other coumarin compounds or phenindione. It is the only one that may be given intravenously or intramuscularly. It has the theoretic advantage of being absorbed completely from the gastrointestinal tract, thus eliminating one factor of variability of response among different patients. However, as with dicumarol, the effects of warfarin sodium are still variable enough among different patients so that no program of fixed doses can be used, and the optimal dose for each patient must be guided by determining prothrombin times. Some physicians consider that a somewhat more rapid and less variable effect can be obtained with warfarin sodium than with dicumarol. Despite conflicting reports, it is doubtful that any one of the coumarin compounds is superior to any other one or that the average physician can employ a particular one with greater ease and still achieve a satisfactory anticoagulant effect. Such effects can be achieved with several agents, and the choice should be left to the individual physician, depending somewhat on his experience and the consistency of his results. If a therapeutic hypoprothrombinemia is established and maintained with any one of these compounds, the patient receives the same protection against thrombosis and encounters the same risk of bleeding.

It must be re-emphasized that different patients vary considerably in their response to coumarin compounds and that any individual patient may vary from time to time in his response to certain fixed doses. Therefore, constant supervision and development of the optimal dose of the compound for each patient are imperative, and this optimal dose can be determined only by trial and error. Daily measurement of the one-stage prothrombin time should be done during the first two or three weeks of treatment at least. If the patient is maintaining a satisfactory response at the end of that time on a fixed daily dose, the frequency of the tests may be gradually decreased to twice or once a week, or even in some instances to once in four weeks. However, the physician must assume the responsibility for doing the test frequently enough to be sure that he is obtaining the proper effect for the individual patient.

Phenindione is not a coumarin compound but it has a similar action and has been recommended by some physicians. Its action resembles that of tromexan in that its effect develops relatively rapidly and disappears rapidly when its administration is discontinued. However, it is more difficult to keep the prothrombin time within the therapeutic range during administration of phenindione than it is with dicumarol or even tromexan.

Long-Term Therapy

For long-term anticoagulant therapy, it has been said that three things are required, namely (1) a co-operative patient, (2) an interested physician and (3) a good laboratory. Physicians who have had long experience in the use of coumarin compounds agree that the one-stage test of prothrombin time is the only satisfactory guide to dosage. I would like to warn against the use of the coagulation time of whole blood for this purpose, although this procedure has been advocated by some investigators. The coagulation time of whole blood is susceptible to numerous errors of technique that cause significant variations in the results. Furthermore, if one depends on this test (even when done by an expert who supposedly has solved all the problems of technique and has done many tests), the anticoagulant effect probably will be excessive in many patients and will result in a considerably increased tendency to bleeding.

In general, a satisfactory therapeutic range of hypoprothrombinemia produced by one of the coumarin compounds is reflected by prothrombin times that are between two and two and one-half times the normal value in seconds for that particular laboratory. These are narrow limits and actually a broader therapeutic zone might be considered as prothrombin times between one and one-half and three times normal, although the narrower range is more desirable, if it is possible to maintain it. However, in discussing the therapeutic range of hypoprothrombinemia, it is best to consider the results of the prothrombin-time test expressed as the degree of prothrombin activity. Normal values for prothrombin time in seconds vary considerably among different laboratories, depending on the exact technique used and particularly on the type of thromboplastin, which is the chief reagent. Many laboratories express results in terms of percentages of prothrombin activity. These should be calculated by interpolation on a dilution curve obtained by plotting the prothrombin times of various dilutions of normal plasma against the percentages of the dilutions. By this method, my colleagues and I consider that the therapeutic range of hypoprothrombinemia for the coumarin compounds is indicated by a prothrombin activity between 10 and 30 per cent. The ideal range is between 15 and 25 per cent.

For long-term coumarin therapy, it is advisable as soon as possible to establish the optimal daily dose for each patient, namely the dose that will maintain the prothrombin time within the therapeutic range. For dicumarol, this dose usually varies between 25 and 125 mg. a day. Sometimes it is necessary to give different doses on alternate days or to give a slightly larger or smaller dose every third or fourth day. Such fine adjustments often are necessary in order to maintain a smooth effect that varies little from day to day as measured by the prothrombin time. Maintenance doses for tromexan usually vary between 300 and 900 mg. a day, and those for warfarin sodium are between 2.5 and 10 mg. a day. The first, or induction, dose of tromexan is 1,200 to 1,500 mg., whereas that of warfarin sodium is 40 to 50 mg.

If the prothrombin time is kept within the therapeutic range, experience has shown that the patient receives maximal protection against thrombosis with minimal danger of hemorrhage. However, an occasional patient may be encountered in whom the stimulus to thrombosis is great and in whom maintenance of the prothrombin time within the therapeutic range is not sufficient to prevent thrombosis. Fortunately, such patients are rare. Also, few patients will experience serious bleeding when the prothrombin time is within the therapeutic range. When such bleeding occurs, it is usually from granulating wounds, ulcers or tumors in the gastrointestinal or urinary tract, or at the site of a contusion or laceration. In most instances of severe bleeding, it will be found that the prothrombin time has been prolonged beyond the therapeutic range. The prothrombin time often may exceed the therapeutic range during the first week of treatment, when the dosage of the drug employed is still in the trial stage. However, in my experience, it is unusual for serious hemorrhage to develop during this first week of treatment, even though the prothrombin time may increase considerably above the therapeutic range, whereas a great deal more danger of hemorrhage exists with such increased prothrombin times during later periods of treatment.

Contraindications

Contraindications to anticoagulant therapy with either heparin or the coumarin compounds are still considered to include blood dyscrasias with a tendency to bleeding, purpura of any type, and recent operations on the brain or spinal cord. In addition, moderate to severe degrees of hepatic or renal insufficiency contraindicate use of the coumarin compounds and phenindione. More than the usual degree of caution must be observed when anticoagulants are given to patients who have ulcerating lesions or granulating wounds and to those who have tubes in various body orifices. Caution should be observed and smaller induction and maintenance doses should be given to patients who have impaired states of nutrition. These include patients who have had surgical operations within the previous five days, those who have been on restricted diets, and those who have gastrointestinal disease, particularly associated with vomiting and diarrhea. Such patients are more than normally sensitive to the coumarin compounds and usually exhibit an excessive response to the doses recommended for patients without nutritional disturbances.

Neutralization of Anticoagulant Effects

For correction of the anticoagulant effect of the coumarin compounds, vitamin K_1 is much more effective than are the water-soluble preparations of menadione. When prothrombin times increase excessively after administration of the usual induction doses of one of the coumarin compounds, this condition may be corrected satisfactorily and the prothrombin time brought back into the therapeutic range by the intravenous administration of menadione sodium bisulfite in a dose of approximately 36 mg. However, when it is necessary to stop the effect of the coumarin compound as rapidly as possible, particularly if serious bleeding has been encountered, it is advisable to use vitamin K_1. This can be given orally and the recommended dose is 250 mg. If the patient is vomiting or has external biliary drainage, it may be given intravenously in the same amount; it is advisable to dilute the material in 300 to 500 ml. of fluid when giving it intravenously. To date there is no evidence that stopping the effect of coumarin compounds by use of these large doses of vitamin K_1 produces hypercoagulability of the blood or a greater tendency to thrombosis than existed prior to administration of the anticoagulant. The large dose of vitamin K_1 is recommended because it will be effective in some cases in which smaller doses, such as 50 mg., will not. Transfusions of whole blood may be advisable when a considerable amount of blood has been lost. In these instances, the transfusions are given to replace blood rather than to replace prothrombin.

Comment

In considering the indications for anticoagulant therapy, the best basic approach is to think of each patient as an individual problem. The likelihood and seriousness of further thrombosis or embolism should be weighed against the difficulty in maintaining and satisfactorily controlling treatment and the possibility of bleeding. In general, short-term anticoagulant therapy is indicated when sural, femoral or iliofemoral thrombophlebitis or pulmonary embolism occurs after surgical operations, childbirth or severe injury, or during the course of infectious diseases. Treatment should be continued for at least two weeks and until the patient has been ambulatory for several days. Similar short-term therapy also is indicated for acute peripheral embolic or thrombotic arterial occlusion. Use of anticoagulants for four to six weeks is indicated in most cases of acute myocardial infarction.

If it can be maintained satisfactorily, long-term anticoagulant therapy usually is indicated in the following conditions: (1) frequently recurring idiopathic thrombophlebitis; (2) chronic auricular fibrillation complicated by intracardiac thrombosis and peripheral or cerebral arterial embolization; (3) recurrent episodes of basilar or carotid insufficiency or cerebral infarcts resulting from thrombosis in the basilar or carotid arterial systems unless such infarcts are large and produce persistent hemiplegia; (4) more than one episode of acute myocardial infarction, particularly if these occur within a few months; (5) frequently recurring peripheral arterial thrombosis and (6) any combination of acute myocardial infarction, peripheral arterial thrombosis and cerebral arterial thrombosis occurring successively during a period of a year or less. Adequate statistical data support the effectiveness of anticoagulant therapy in most of the afore-mentioned conditions. For acute peripheral arterial or venous thrombosis and pulmonary

(Continued on Page 272)

Diagnosis and Treatment of Local Edema of Lower Extremities

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SWELLING of the lower extremities is a common presenting complaint of the patient to the physician. The patient often fears that the swelling is caused by "dropsy," a fear that induces pronounced but usually needless anxiety in the patient. In addition, a swollen extremity is cosmetically unattractive, a fact that often brings the patient to the physician. Despite patients' fears, the majority of edematous extremities are not associated with serious cardiac, renal or hepatic disease but instead are brought about by local factors and often can be relieved by local treatment. Some of these local factors are summarized in Table I, together with some additional causes of edema. Although each of the local conditions that cause edema of an extremity can, and occasionally does, affect the upper extremities, by far the commonest site of occurrence is in the lower extremities.

Exclusion of the more central or systemic origins of edema, such as congestive heart failure, nephritis or the nephrotic syndrome, cirrhosis or endocrine disorders, leaves five main causative factors for swelling of an extremity, namely physiologic edema, thrombophlebitis, chronic venous insufficiency, lymphedema and lipedema.

Physiologic Edema

Types of edema that may be considered "normal," or "physiologic," are gravitational edema and premenstrual edema. The normal lower extremity that is inactive and dependent often has an accumulation of fluid because of increased hydrostatic pressure. This is commonly seen in persons who travel long distances in trains or automobiles or who sit for hours without activity. People who stand or sit at their work hour after hour without walking about may be similarly affected. Rarely, such persons may be almost incapacitated by the accumulation of edema; however, more often the problem is one of cosmetic deformity or mental anguish rather than of true incapacity. Reassurance of the patient that this swelling does not represent serious disease, and the use of elastic support and, occasionally, a restricted intake of salt often give these patients help. Edema occurring premenstrually is attributable in part to retention of sodium and water. The precise hormonal abnormality is not known but restriction of fluid and salt sometimes produces remarkable, although rarely complete, relief. These patients do not have edema during the remainder of the menstrual cycle.

TABLE I. CLASSIFICATION OF EDEMA OF LOWER EXTREMITIES

Primary, or idiopathic
- Gravitational and physiologic edema
- Lipedema
- Lymphedema

Secondary, or symptomatic
- *Localized or regional disease*
  - Thrombophlebitis
  - Lymphedema
  - Edema of venous stasis
  - Angioneurotic edema
  - Myxedema (pretibial myxedema associated with exophthalmic goiter)
- *Systemic disease*
  - Renal disease: nephritis and nephrosis
  - Myocardial disease: heart failure
  - Hepatic disease: decompensated cirrhosis
  - Endocrine disease: hyperthyroidism; myxedema; aldosteronism; Cushing's syndrome

Treatment-induced edema
- High-salt intake
- Salt-retaining hormones: desoxycorticosterone; cortisone; corticotropin; testosterone; aldosterone; antidiuretic hormone
- Rauwolfia preparations

Thrombophlebitis

Thrombophlebitis involving only the superficial venous system does not cause edema; however, thrombophlebitis involving the deep system of veins is usually associated with a relatively acute onset of unilateral edema, pain or tenderness in the involved extremity, and the other classic signs of deep venous thrombosis (Fig. 1). With elevation of the leg and use of anticoagulants, the edema subsides within a matter of days. After the signs of acute inflammation subside and the extremity is free of edema, the patient with throm-

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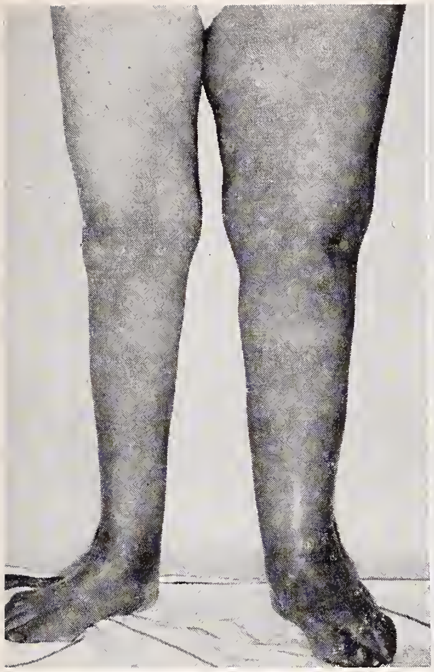

Fig. 1. Left iliofemoral thrombophlebitis.

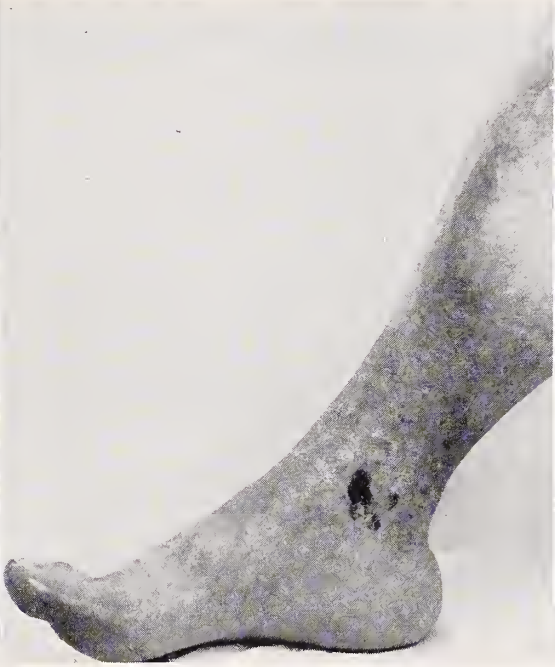

Fig. 2. Chronic venous insufficiency with stasis dermatitis and ulceration.

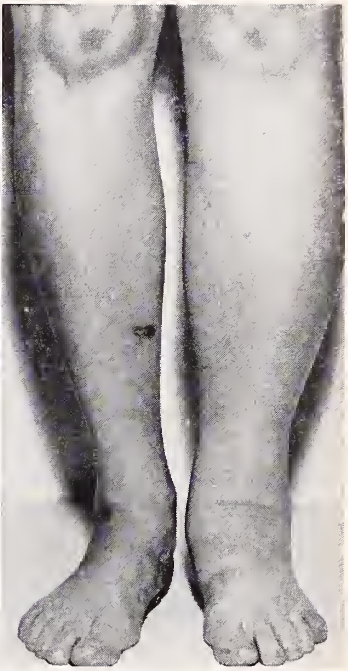

Fig. 3. Primary lymphedema of left leg, showing "pigskin" appearance.

bophlebitis of the calf or of an iliofemoral distribution should have two elastic bandages applied to the leg before he gets out of bed. These supports should include only that part of the leg from the instep to the infrapatellar region, since it is almost impossible to have a bandage stay in place above the knee. All patients who have iliofemoral thrombophlebitis and most patients with sural thrombophlebitis will have dependent edema immediately after they become ambulatory, and it is unwise to wait to see how much swelling may develop. The patient may become free of edema when adequate elastic support is used for a period of weeks or months.

Chronic Venous Insufficiency

If destruction of the venous valves is extensive and recanalization of the thrombosed vein is inadequate after thrombophlebitis, a tendency for chronic swelling of the extremity may be present. This is chronic venous insufficiency, which most often results from inadequate treatment of the patient during the acute episode of phlebitis and in the period of weeks or months immediately after active thrombophlebitis.

Edema secondary to chronic venous insufficiency may result from involvement of the superficial or the deep venous system or both. The basic defect in patients with edema caused by chronic venous insufficiency is the absence of competent valves, which leads to a pronounced increase in venous pressure in the leg. Normally, the venous pressure at the ankle decreases rapidly with exercise and slowly returns to normal. In a vein without competent valves, the venous pressure does not decrease as much as it does in normal situations and it returns more rapidly to the level present before exercise. With this persistence of increased venous pressure, transudation of fluid is promoted and edema results. An additional factor in the formation of edema may be the relative stasis of blood in the incompetent veins, leading to capillary damage and further promotion of edema. As long as the extremity is drained of all the edema fluid before application of elastic bandages, adequate elastic support almost always controls the edema. Any support is adequate as long as it controls the edema; in my experience, elastic bandages and, occasionally, rubber bandages are needed to control the edema associated with chronic venous insufficiency. However, elastic stockings may control the edema produced by varicose veins.

Some patients with the postphlebitic syndrome or chronic venous insufficiency will have chronic indurative cellulitis and some will go on to have periodic lymphangitis and subsequent lymphatic obstruction, leading to localized or regional lym-

TABLE II. CLASSIFICATION OF LYMPHEDEMA

- *Noninflammatory*
  - *Primary*
    - Praecox
    - Congenital
      - Simple
      - Milroy's (familial)
  - *Secondary*
    - Malignant occlusion
    - Surgical removal of lymph nodes
    - Pressure
    - X-ray and radium therapy
- *Inflammatory*
  - *Primary* (single episodes, recurrent acute or chronic episodes)
  - *Secondary* (single episodes, recurrent acute or chronic episodes)
    - Venous stasis
    - Trichophytosis
    - Systemic disease
    - Local tissue injury

phedema; more frequent than these complications are the usual sequelae of chronic venous insufficiency, namely stasis pigmentation, dermatitis and ulceration (Fig. 2). The majority of these complications can be prevented by the avoidance of chronic edema in an extremity. If the patients are alerted to these late complications and understand that elastic support is used for the purpose of avoiding chronic edema in order to prevent subsequent ulceration, cellulitis and lymphangitis, they will be more likely to be careful about using elastic bandages or stockings.

Lymphedema

Lymphedema is a relatively uncommon condition that is confused most often with chronic venous insufficiency; if the lymphedema is acute in onset, it may be confused with deep venous thrombosis. Although there are many causes of lymphedema (Table II), there appears to be a common mechanism of production.

Stasis of lymph occurs initially because of obstruction to the normal flow of lymph. This obstruction may be caused by inflammation, congenital lymphangiectasis or malignant invasion. With subsequent thrombosis of lymphatic vessels, proliferation of fibroblasts occurs in the lymph, which is an excellent culture medium for tissue, and with the destruction of valves in the lymphatic vessels, new but incompetent lymph channels appear and the same cycle recurs. The region eventually becomes edematous without signs of venous disease. The lymphatic systems of the leg are divided into deep and superficial portions by the deep muscular fascia. It is the superficial system that becomes involved mainly in lymphedema; the skin is thickened, and the hair follicles become prominent, giving the appearance commonly referred to as "pigskin" (Fig. 3). Early in lymphedema, the swelling is soft and pittable, and it often recedes completely on elevation of the extremity. Later, the tissues become more and more indurated and less of the swelling disappears with elevation; some tissue hypertrophy occurs, which also increases the size of the leg. The usual site for lymphedema is in one lower extremity, including the foot, although both legs may be involved. In the congenital type, an arm or even the face may be lymphedematous.

In treating lymphedema, it is important to attempt to reduce the swelling to a minimum by continuous twenty-four-hour elevation of the leg, which is done best in the hospital. After one to three days of elevation, all the swelling that can be eliminated usually will have left. The patient is then instructed in the proper application and use of an adequate elastic support. A Pará gum-rubber bandage is the most effective type of support, although it is heavy and often uncomfortable, especially in warm weather. A lisle stocking, worn under the rubber bandage, gives some protection against dermatitis and skin irritation from excessive perspiration. With continued use of this support, the extremity will not continue to enlarge or its rate of enlargement may be slowed. The patient should be warned that the lymphedematous extremity is a frequent site for infection or lymphangitis, that such infection may produce further enlargement, and that prompt antibiotic treatment of the infection is necessary. Trichophytosis should be treated and controlled, since such fungous infections may provide sites of entry for bacteria. The surgical treatment of lymphedema consists in wide excision of lymphedematous subcutaneous tissue. Its use is reserved for massively enlarged limbs, and the patient who elects surgical intervention often trades a swollen limb for a badly scarred and still abnormal-appearing extremity.

Again, as in chronic venous insufficiency, the patient should be thoroughly instructed and oriented as to the need for adequate elastic support and the methods of prevention and treatment of lymphangitis. Even with enthusiastic encouragement by the physician, a large number of patients discard their bandages and their legs progressively enlarge, becoming subject to the complications of untreated lymphedema.

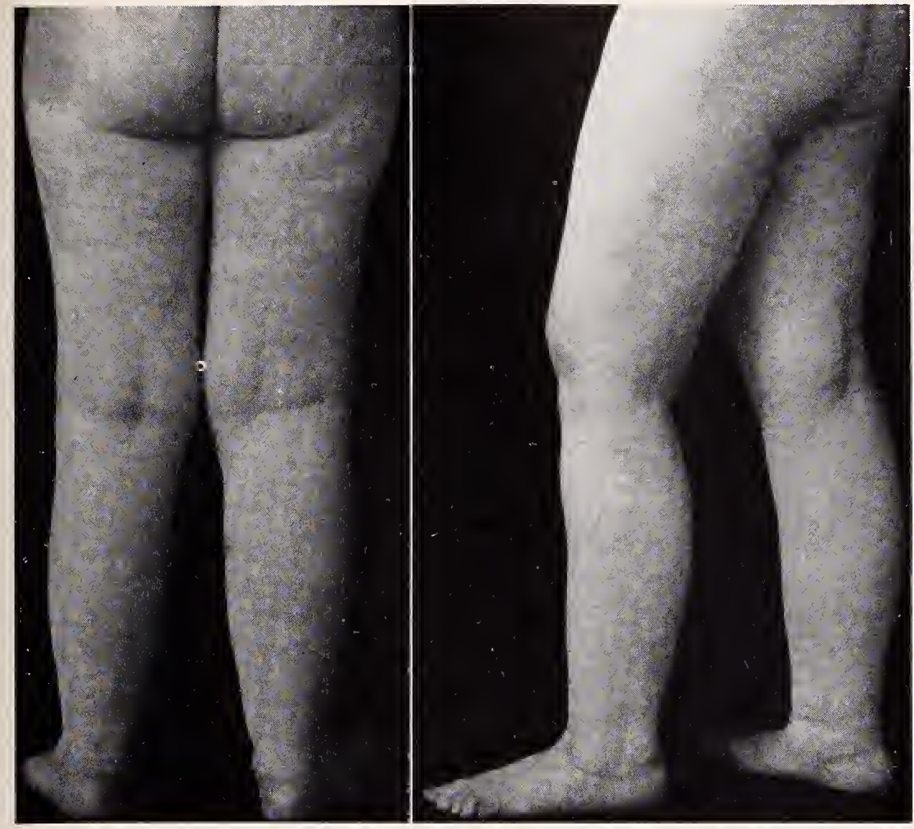

Fig. 4. Typical appearance in lipedema. Note absence of involvement of feet.

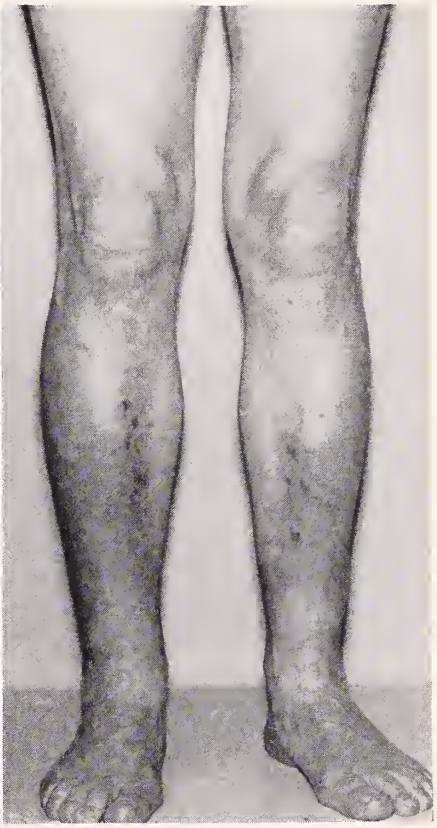

Fig. 5. Pretibial myxedema.

Lipedema

Lipedema means the deposition of excessive amounts of fat in the legs, with secondary orthostatic edema. This condition is almost always bilateral and affects women almost exclusively. A family history of similar fatty swelling may be present. The cause of this unusual fatty accumulation is not known. It may be present in childhood and may be exaggerated by recent gain in weight, yet reduction of weight does not appear to alter the basic appearance of the legs. The upper part of the trunk and the upper extremities usually appear normal, with an accumulation of fat about the hips and extending into the thighs and legs but not involving the feet (Fig. 4). The skin is normal in appearance but is often hypersensitive. Pitting edema usually cannot be demonstrated, because the collections of extracellular fluid lie in the interstices between fatty deposits; however, some patients may note pitting edema especially in warm weather or when they are on their feet for long periods.

Rarely, lipedema may involve both arms.

The treatment consists mainly of reassuring the patient that this condition does not represent any serious disease. Elastic supports occasionally help to prevent accumulation of fluid, but the patient often cannot tolerate tight bandages or stockings because the skin is tender.

Other Types of Local Edema

Any patient who manifests edema of obscure cause or who notes an unexplainable increase in swelling of the leg should be closely questioned as to the ingestion of medicaments, because drug-induced edema may explain this swelling. The Rauwolfia drugs, hydralazine (apresoline) hydrochloride and cortisone or other adrenal steroids are common offenders in this regard.

An unusual type of local edema may occur only in the pretibial region or on the dorsum of the foot. This is termed "localized" or "pretibial" myxedema (Fig. 5). However, this terminology is poor, since the condition bears no relationship to the presence or absence of generalized myxedema; instead, it is always seen in patients with exophthalmic goiter. It is usually a self-limited process that disappears over the course of years. Such swelling might be mistaken for lipedema, but the peculiar localization to the pretibial region and the foot is not seen in lipedema.

(Summary on Page 277)

A New Concept in the Surgical Treatment of Aortic Insufficiency

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ALTHOUGH stenotic lesions of the aortic valve were surgically attacked as early as 1913 by Tuffier,[1] who attempted dilatation by invagination of the aortic wall, little was done toward the sugical correction of aortic regurgitation until Campbell[2] in 1950 and Hufnagel[3] in 1951 introduced prosthetic valves in the descending portion of the aorta. Other methods tried experimentally and clinically for the control of aortic regurgitation include transplantation of homologous aortic valves,[4,5] injection of sclerosing solutions around the aortic annulus,[6] pericardial pedicles[6] and vein grafts,[5] organic prostheses,[7] and circumferential, pursing sutures of the aortic annulus.[6,8] Supracoronary and subcoronary prosthetic valves have also been described.[6,9,10,11] Although placement of a prosthetic valve in the descending aorta has received extensive clinical trial by Hufnagel, limitations of the method are: (1) the partial control of regurgitation because of the distal location of the artificial valve; (2) the relation of the coronary arteries to the new aortic valve is reversed; (3) a permanent foreign body is introduced into the blood stream with the possible sequelae of trauma to blood cellular elements and thrombus formation. The circumclusion technique introduced by Bailey was abandoned by him after forty-three clinical trials.

Inasmuch as acquired forms of aortic insufficiency are characterized by a loss of valve substance and dilatation of the aortic annulus,[12] an operation was designed to alter the pathologic anatomy, namely, to permanently reduce by one third the circumference of the dilated aortic valve ring by totally excising the non-coronary aortic cusp and its corresponding segment of aortic wall, converting the tricuspid aortic valve to a bicuspid valve (Fig. 1). In so doing, improved coaptation of the two remaining damaged coronary cusps is achieved. Accordingly, the normal relationship between the reconstructed aortic valve and coronary arteries is retained and no foreign body is introduced into the vascular system.

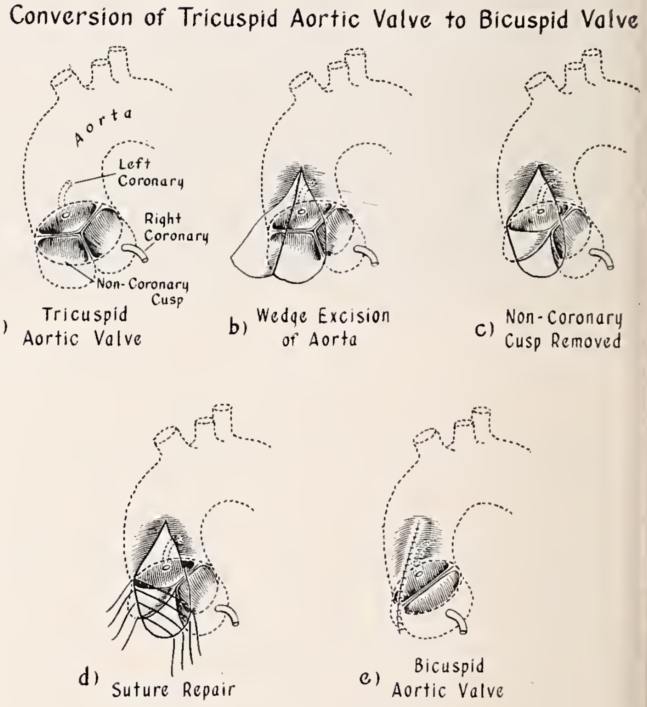
Conversion of Tricuspid Aortic Valve to Bicuspid Valve
Aorta
Left Coronary
Right Coronary
Non-Coronary Cusp
a) Tricuspid Aortic Valve
b) Wedge Excision of Aorta
c) Non-Coronary Cusp Removed
d) Suture Repair
e) Bicuspid Aortic Valve

Fig. 1. Schematic drawings showing method of converting the tricuspid aortic valve to a bicuspid valve by total excision of the non-coronary aortic cusp and a corresponding segment of the aortic wall. Procedures were done under direct vision employing a pump-oxygenator and elective cardiac arrest.

In order to test this concept, experimental studies were performed on dogs to determine whether or not conversion of the tricuspid aortic valve to a bicuspid valve was compatible with survival, and secondly to determine whether or not significant degrees of dynamic aortic stenosis and/or aortic insufficiency were produced. In effect, a severe degree of aortic insufficiency is produced by total excision of the non-coronary

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aortic cusp and this is surgically corrected by the technique described. A defect of more than one third of the posterior cusp has been found intolerable in experimental animals.[13] Operative procedures were performed under direct vision with cardiac by-pass employing an extracorporeal pump-oxygenator[14] and elective cardiac arrest.[15]

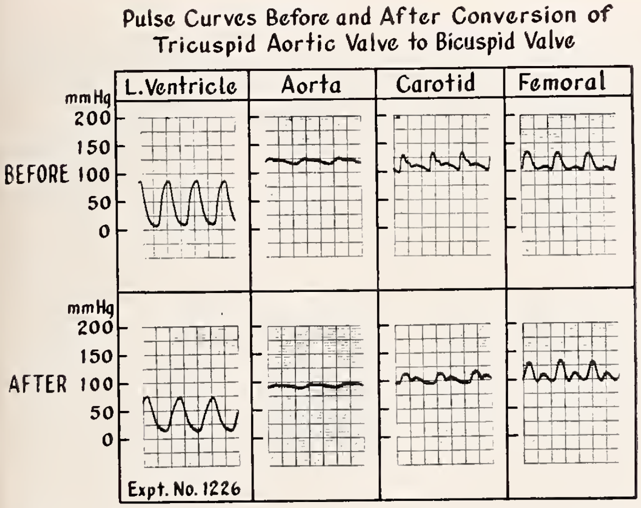

Fig. 2. Arterial pulse curves taken before and after conversion of the tricuspid aortic valve to a bicuspid valve show slight elevation of the end-diastolic pressure in the left ventricle, absence of a gradient across the aortic valve and dicrotic notches and dicrotic waves in the aortic, carotid, and femoral pulse tracings. The dicrotic notch and wave are representative of aortic valve closure.

Results

In twenty consecutive studies there were eleven acute survivors. Seven animals are long-term survivals (four to six months). Additional chronic survivors not included in this analysis have been obtained, however.

During our early experience with the method seven of nine acute postoperative deaths were due to severe dynamic aortic insufficiency as evidenced by absence of the dicrotic notches and dicrotic waves in the arterial pulse curves in the postoperative period. The dicrotic notch and dicrotic wave are representative of aortic valve closure.[16-19] A more deliberate coaptation of the newly formed posterior commissure of the bicuspid valve in the last nine of the twenty studies was attended with consistent post operative dicrotic arterial pulse curves and improved animal survival, seven acute survivors and two preventable deaths (hemothorax and tracheal obstruction). Previous postoperative studies[20] including analyses of arterial pulse curves (Fig. 2), measurements of the duration of the ejection phase of systole, retrograde aortography (Fig. 3), and transvalvular pressure determinations showed no significant degrees of aortic stenosis and/or aortic insufficiency in the surviving animals.

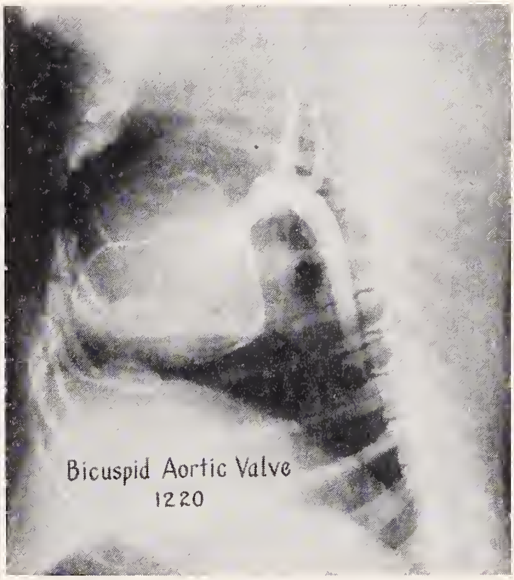

Fig. 3. Postoperative retrograde aortogram showing: (a) bicuspid nature of a aortic valve, (b) surgical narrowing of aorta, (c) coronary artery filling, (d) absence of dye in the left ventricle. Arterial pulse curves showed dicrotic notches and dicrotic waves.

To determine the feasibility of this operative procedure in clinical aortic regurgitation a series of photographs were prepared from an autopsy specimen obtained from a patient that succumbed of acquired aortic insufficiency. These are shown in Figs. 4, 5, 6, 7 before and after conversion of the tricuspid aortic valve to a bicuspid valve. The anatomic reduction of the regurgitant site is apparent following the operative procedure.

Other preliminary investigations showed that if the aortic wall was reduced by one third without excision of the corresponding non-coronary cusp, there is, in effect, a herniation of the non-coronary cusp below the two coronary cusps in which case it does not appear to contribute to the function of the refashioned valve and probably detracts from it.

Isolated disease of the non-coronary cusp (as following trauma or congenital perforation, for example) seemingly would be suited for this type repair. Possible clinical application for other acquired regurgitant pulmonic lesions is suggested.

Summary

A new concept in the surgical treatment of aortic insufficiency is introduced. The surgical method consists of plastic revision of the tricuspid aortic valve to a bicuspid valve by total excision of the noncoronary aortic cusp. Experimental data and a preliminary pathologic study support the feasibility of the method.

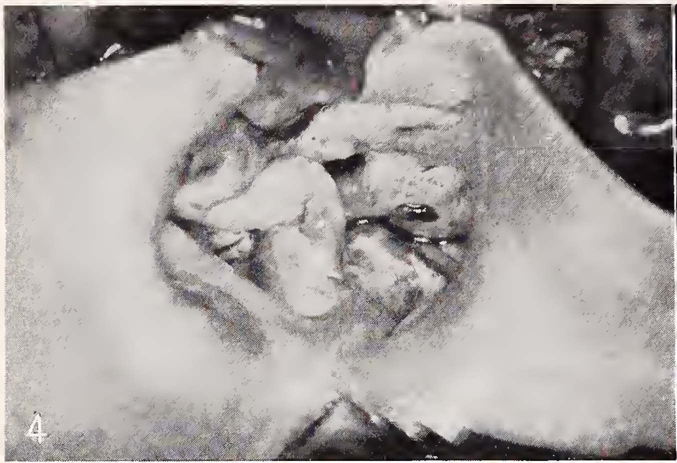

Fig. 4. An autopsy specimen from a patient dying of aortic regurgitation shows the insufficient aortic valve viewed from above and posteriorly. Note the distortion, thickening and shortening of the aortic cusps. The central defect causing aortic regurgitation is evident. The loss of cusp substance precludes complete coaptation. Dilatation of the aortic ring is not a conspicuous feature of this specimen.

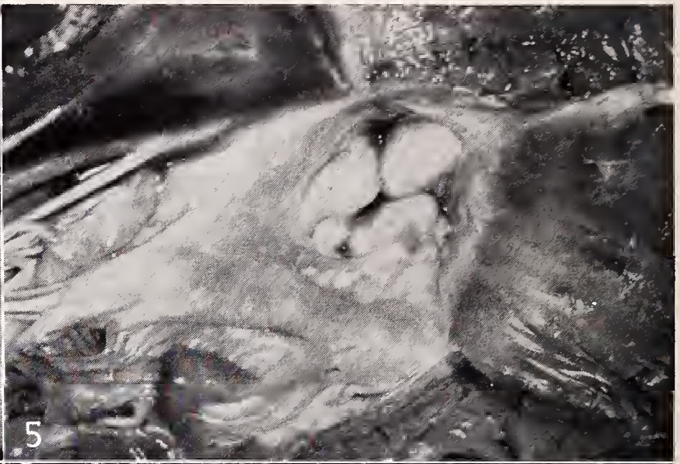

Fig. 5. Insufficient aortic valve viewed from below, the ventricular side, showing the central defect causing regurgitation. The posterior, non-coronary cusp occupies the lower part of the valve orifice. A rim of calcification is noted on the right.

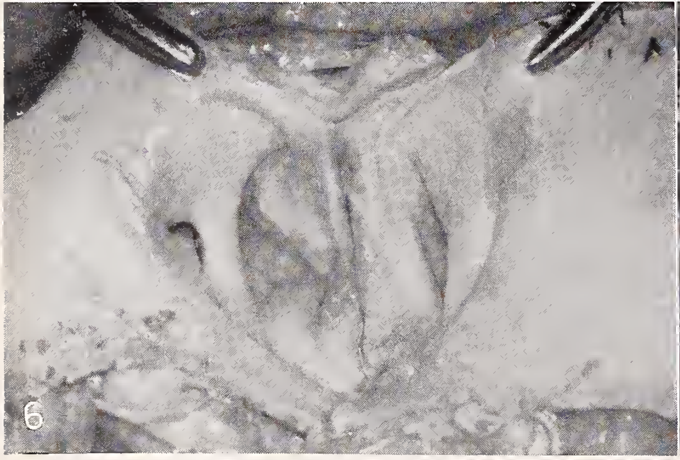

Fig. 6. The incompetent aortic valve seen in Figs. 4, 5 is viewed from above following plastic revision with total excision of the non-coronary aortic cusp. The valve is now bicuspid. The circumference has been reduced by one-third. The remaining coronary aortic cusps appear to coapt well. The left coronary ostium is visualized to the left.

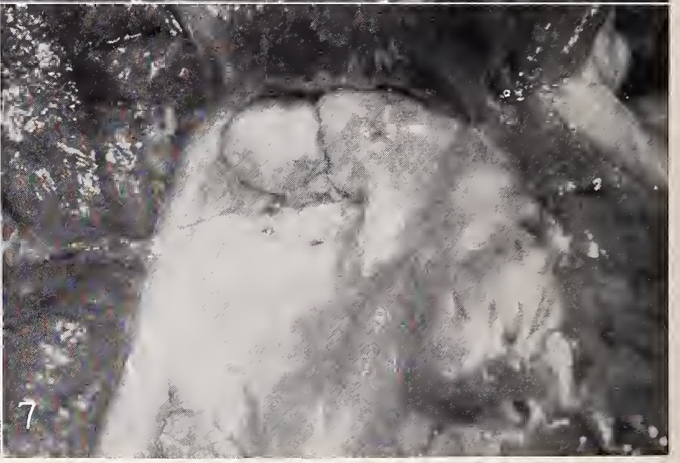

Fig. 7. The incompetent aortic valve seen in Figs. 4, 5 is viewed from below, ventricular side, following plastic revision. The non-coronary aortic cusp has been excised. The newly formed bicuspid valve appears to coapt well. Sutures in the line of repair are evident.

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A New Antirheumatic Steroid

Preliminary Report

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ALMOST immediately following the demonstration of the physiologic effects of cortisone in human patients,[1] and the subsequent correlation of the chemical structure of related steroid compounds with their physiologic action,[2,3] a sustained effort has been going on in many centers to develop and test compounds of maximal therapeutic effectiveness but with a minimum of the adverse side effects.[4] The major "desirable" effect of these compounds is their anti-inflammatory action. This property, which has been referred to by Selye as the anti-phlogistic[5] effect, appears to be non-specific regarding the type of inflammation affected and has extended the clinical usefulness of the compounds to such diverse disorders as rheumatoid arthritis, ulcerative colitis, iritis, pemphigus and certain specific infectious diseases.[6,7]

The major "undesirable" effects relate to (1) pituitary and adrenal cortex suppression, (2) Sodium retention and potassium loss—the mineral-corticoid effect, (3) increased protein catabolism, or the anti-anabolic effect which may explain the osteoporosis, the altered reaction of tissue to healing and infection,[8] and the high incidence of peptic ulcer, and (4) collagen vascular disease[9] or so-called hypercortisonism.[10] Thus far, the only undesirable effect that has been significantly altered with the newer steroids has been the marked lessening of sodium retention and potassium loss with the delta-1 steroids prednisone and prednisolone. However, along with the increased antirheumatic potency of these new compounds, a comparable intensification of the glucocorticoid and other effects has occurred and the major hazards of steroid therapy remain.

Method

We wish to report our clinical findings in a group of ambulatory rheumatoid arthritic patients who were studied critically in an attempt to compare a new steroid, 6-methylprednisolone (or $\Delta^{1,4}$ 6 α-methylhydrocortisone with the more usually employed corticoids.

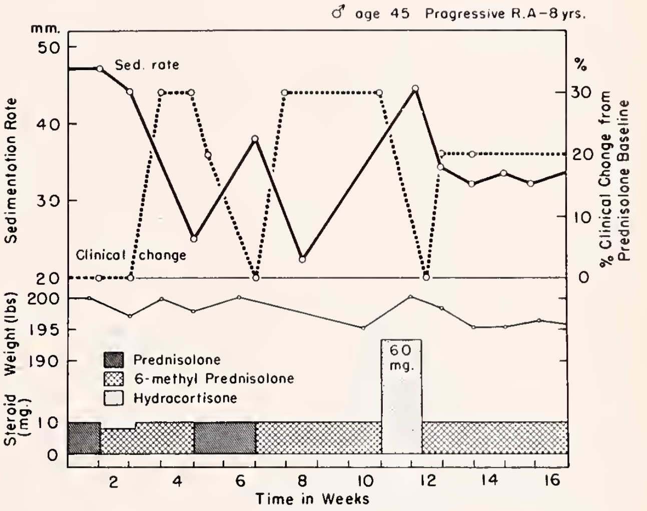

Fig. 1. Comparison of corticoids used in treating a patient with rheumatoid arthritis.

Twenty patients who were being maintained on prednisolone were given 6-methylprednisolone in doses of 2 to 10 mg. per day. The usual procedure was to vary the dosage of the new drug until a clinical level of control was obtained that was equal to that which the patient experienced on his maintenance prednisolone dosage. The patient was them returned to the original prednisolone medication. In eighteen cases, the new medication was given again as a check on the first trial; in several patients, more than two trials were given. Objective joint examinations were performed with measurement of swelling, tenderness and range of motion. The patients were seen at weekly intervals and recordings were made of

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The 6-methylprednisolone used in this study was supplied as Medrol by Dr. H. C. Peltier of the Upjohn Company, Kalamazoo, Michigan.

weight, blood pressure, hemoglobin, and sedimentation rate.

Results

The graphic record of one patient is presented in Figure 1. In Table I, the findings in the twenty patients are summarized. Two patients required the same dosage of 6-methylprednisolone as prednisolone for a given degree of improvement. The remaining eighteen patients, or 90 per cent, required only 50 to 75 per cent of the prednisolone dosage. In seven cases, the ratio of prednisolone to 6-methylprednisolone was 4:3, in nine cases it was 3:2 and in two cases it was 2:1.

TABLE I. 6 METHYLPREDNISOLONE (M) FINDINGS IN TWENTY CASES
As compared with Prednisolone (P)

Case	Duration of Therapy in Weeks	Relative Anti-Rheumatic Potency	Weight Change and Other Effects
1	5	7.5 mg. P= 5 mg. M	Loss 4 lbs.
2	14	15 mg. P=10 mg. M	Loss 2 to 5 lbs; less GI distress.
3	10	10 mg. P=7.5 mg. M	Loss 2 to 4 lbs; better appetite; "more drive."
4	10	7.5 mg. P= 5 mg. M	Loss 3 to 7 lbs; initial epigastric distress.
5	4	6 mg. P= 5 mg. M	Loss 2 lbs; severe headache on second trial.
6	6	6 mg. P= 4 mg. M	Weight same; "more alert."
7	6	4 mg. P= 2 mg. M	Loss 1½ to 4 lbs.
8	4	5 mg. P= 4 mg. M	Loss 4½ lbs.
9	4	6 mg. P= 4 mg. M	Loss 1 lb.
10	2	6 mg. P= 4 mg. M	Loss 2 lbs.
11	1	7.5 mg. P= 5 mg. M	Loss 1 lb.
12	4	8 mg. P= 8 mg. M	Loss ¾ lb.
13	2	5 mg. P= 4 mg. M	Loss ¾ lb.
14	4	5 mg. P= 5 mg. M	Loss 3 lbs.
15	3	5 mg. P= 4 mg. M	Loss 5 lbs.
16	1	5 mg. P= 4 mg. M	Loss ½ lb.
17	3	6 mg. P= 4 mg. M	Loss 3 lbs; "quicker effect and more even."
18	2	5 mg. P= 3 mg. M	Weight same.
19	6	6 mg. P= 4 mg. M	Loss 3 lbs.
20	3	6 mg. P= 5 mg. M	Weight same.

Three patients showed no weight difference on the two medications and two patients lost less than 1 pound in body weight on taking 6-methylprednisolone. Nine patients lost from 1 to 3 pounds and six patients lost from 3 to 5 pounds. Most patients were aware of increased urination, and several who had clinical edema noticed less ankle-foot swelling. The weight loss usually occurred in the first three days but in some patients a slower and steady weight loss was observed. Except for the diuresis, no other striking clinical differences were observed. Several patients with facial "mooning" retained this feature on 6-methylprednisolone. The period of observation has been too short to draw any conclusions concerning the other major side effects of this new compound.

Comment

It has been postulated that alterations in metabolism of the steroid compounds may account for the altered biological effects. Liddle and Richard reported that 2-methylhydrocortisone is removed from the plasma more slowly than hydrocortisone.[11] Prednisolone is excreted in the urine unchanged to a much greater extent than is hydrocortisone which occurs in the urine mainly as a reduced and conjugated metabolite.[12] An effort to assess this alteration in metabolism was made by the intravenous administration of hydro-

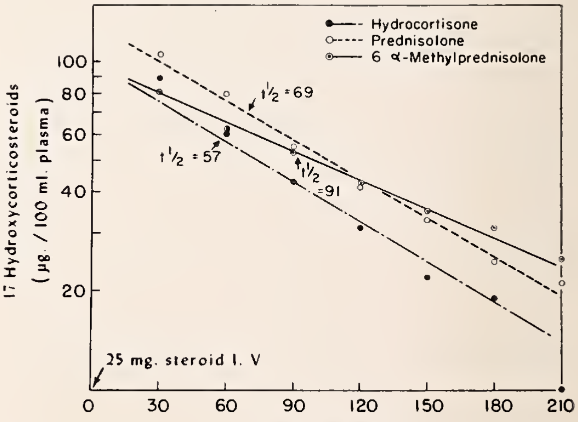

Fig. 2. Plasma biological half-lives of hydrocortisone, prednisolone and 6-methylprednisolone (Medrol) in the dog.

cortisone, prednisolone and 6-methylprednisolone in dogs. After the injection of 25 mg. of these steroids, blood specimens were obtained at thirty-minute intervals and analyzed for 17-hydroxycorticosteroids by the method of Silber and Porter. The mean plasma biological half-lives were calculated from these data. The plasma biological half-life for hydrocortisone was fifty-seven minutes; for prednisolone, sixty-eight minutes; and for 6-methylprednisolone, ninety-one minutes as, graphed in Figure 2. The prolonged plasma biological half-life of 6-methylprednisolone can be correlated with recent studies which show that this compound is resistant to attack by liver enzymes and hence is slowly metabolized as compared with the parent steroids.[13] Reported experimental data in rats indicates that the new compound, when compared with prednisolone, was two times as potent on the anti-inflammatory test, about three times as potent on glycogen deposition assay, and in contrast to prednisolone this steroid acted as a diuretic and natri-uretic agent.[14]

Summary

A new antirheumatic steroid, 6-methylprednisolone, was compared with prednisolone in twenty patients with rheumatoid arthritis. The new compound appears to be about 25 per cent more potent than prednisolone and has mild diuretic properties. No other striking differences from the usually employed steroids were seen in this preliminary study. The diminished rate of removal of 6-methylprednisolone from the plasma of dogs, as compared with prednisolone and hydrocortisone, may relate to the enhanced clinical effectiveness observed.

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PERIPHERAL VASCULAR DISEASES

(Continued from Page 240)

discussion on a note of pessimism. However, I have lived through the era of intermittent suction and pressure and intermittent venous occlusion. The machines used for these purposes now belong quite properly in a medical museum. The medical treatment of hypertension, the proper use of anticoagulants, and the development of arterial surgery are great and thrilling accomplishments, as are other methods of treatment which I have mentioned. When one views the general field of peripheral vascular diseases, he can only be optimistic.

Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

INFANT FEEDING

The Evaporated Milk Chapter

That more than two-thirds of the babies born in U. S. hospitals are discharged on a bottle formula of some kind was disclosed in a recent survey. In these formulas evaporated milk is being prescribed more than all other forms of cow's milk combined, including the special proprietary infant feeding preparations.

It was not always thus. Prior to 1929, the medical literature contained infrequent reports on the use of evaporated milk in infant feeding. In that year, two great investigators in infant nutrition, independently and coincidentally, published the results of their studies on large numbers of babies, Brennemann, in the February 2 issue of the *Journal of The American Medical Association* and Marriott in the March issue of *Archives of Pediatrics.*

Brennemann reported his observations in particularly glowing terms.—"The result was all that has been claimed and all that could be hoped for, and again such as I had never seen before with any other food. . . . The gain in weight was better than before—sometimes truly amazing—and, I feel sure, as good as if the babies had been on breast milk." Marriott reached similar conclusions based on evaporated milk formula feeding to 752 young infants.

Doctor Brennemann had long been concerned with the curd problem when ordinary cow's milk was used in the feeding formula. He observed that with evaporated milk, under conditions simulating those existing in the infant stomach, the casein coagulates not in a cheese-like mass, but in a soft semi-fluid condition comparable to the protein coagulum from human milk. He believed that this reduction in curd tension removed the most important source of difficulty that arises from the ingestion of formulas prepared from cow's milk; and this is generally accepted in the medical profession at this time. Certainly, all products that are promoted as the basis for formula construction stress the point of curd tension.

During the 1930's, numerous other clinics conducted infant feeding studies, comparing the nutriture of infants on evaporated milk formulas with other forms of cow's milk and even with human milk. Mineral and nitrogen retentions were determined. Fat digestibility experiments were performed, and the growing list of vitamins, to which so much attention was being given during that period, came under the scrutiny of the laboratory technologists.

As expected, they found that some of the vitamins were reduced appreciably percentagewise in the technique of converting raw cow's milk into evaporated milk, but there was no loss that would indicate the institution of precautionary measures other than had been practiced before, such as the routine administration of vitamins C and D. On the other hand, the researchers observed that certain of the nutrients were even better utilized from evaporated milk formulas than from formulas prepared from other forms of milk. The apprehension which had existed before, which actually materialized as a prejudice against "canned milk" was gradually dispelled.

In the process of producing evaporated milk from raw cow's milk sufficient heat is applied to protect it against spoilage. This is known to be the principle factor that brings about the favorable change in the casein. Homogenization also is a factor in lowering curd tension, though a minor one.

Actually the homogenization process is applied for the purpose of reducing the fat globules to such size that they do not "cream off" during the shelf life of the can of milk. But it happens also to add a nutritional advantage; as the milk fat in this highly homogenized form presents a greater surface to the fat digestive enzymes. The milk fat becomes more readily digested, an effect especially important in difficult feeding cases.

The ease with which the infant digests and metabolizes the nutrients in the milk has very likely contributed to the success that has been experienced in many medical centers with formulas prepared by merely diluting the milk with water, and without addition of carbohydrate.

Among other factors that have advanced the use of evaporated milk in the preparation of the formula have been the observed reduced allergenicity of the protein composite, especially the whey proteins, and the universal fortification of the product with vitamin D. But each of these subjects could easily be made a separate "chapter" in the infant feeding story. Also there is an extensive literature reporting the widespread and successful use of evaporated milk as the basis for special formulas for prematures.

That the advantageous feeding of evaporated milk is not limited to the neonatal period has been demonstrated in a number of studies where it has been the single form of milk given children for drinking throughout the growing years. This is of particular consequence in areas where there is an inadequate supply of good quality low cost market milk.

And there are a number of practical reasons why the medical profession has accepted evaporated milk as a desirable form of cow's milk for child feeding—uniformity of product from day to day and city to city, and its universal availability at relatively low cost are among the contributing factors. Also important to many physicians is the fact that it lends itself to individual formula modification.

In the United States and in certain other parts of the world there has been a striking reduction in infant mortality in recent years. The growing use of evaporated milk, especially vitamin D evaporated milk has been credited with being a contributing factor. Other factors of equally great importance, of course, have been—improved medical care, better home sanitation and an increasing understanding through research of infant digestion and metabolism.

Present day medical literature contains fewer contributions on routine infant feeding techniques than formerly. The factors that make for successful formula feeding are quite well established. Infant disorders are found more likely to arise from causes other than food. However, in the feeding of the disordered infant mechanism there are gaps in our present knowledge. The cow's milk formula does differ from human milk in a number of respects—more protein, more electrolyte, a different fat composition and Ca:P ratio. Just how significant are these differences in feeding the sick baby is quite properly the subject of intensive clinical study.

It is well that the doctor does not need to give as much attention to the feeding of the normal baby as he did thirty years ago. Physicians can now become really "physicians for the young of the race instead of highly paid nannies," as Dr. Alton Goldbloom recently put it.

Frank E. Rice, Ph.D.

TRENDS IN MEDICINE

Let me assume for the moment that our fictional patient has completed his check-up. His doctor, an impossible paragon if he has investigated all facets of our patient's physical, mental and spiritual health, sums up:

> "There is not anything fundamentally wrong with you. Your heart, lungs, intestinal and urinary tracts are in excellent condition. Your electrocardiogram is normal as are your numerous blood tests. True, your blood pressure has been somewhat higher than the average for your age ever since your recent promotion, and I cannot approve of your working so intensely. I do appreciate that you will gain more weight if you stop smoking, but this is not a sound reason for your smoking to excess. The fact that you feel you need the three drinks before dinner indicates more tension than you admit, as does your restless, non-refreshing sleep. I was sorry to hear of your father's untimely death from a stroke, and I am certain that your brother's coronary worries you. These two vascular accidents in the male side of your family do not mean that you will have one too, but they should be added incentives to keep you in the healthiest possible condition. I strongly urge that you lose about 15 pounds and reduce your tobacco consumption by half. I also urge that you get regular exercise and develop other interests besides your work. In fact the best way to start on this program would be to take several weeks' vacation at once."

This is not an atypical summary of an intelligent physical appraisal, nor is it lacking except in concrete recommendations. Partly because I myself have made this speech so often in my practice without being able to go further I was induced to leave my practice for the Aspen project.

The Aspen Institute program is not a new idea, for the basic principles go back to Greek and Roman days. Nor is there any mystery or fetishism in the program. It is neither an intellectual bull-session nor a bake and massage center. Rather it is an effort to combine organized conversation with physical rehabilitation. Both mind and body need stretching beyond the threshold of stimulation if they are to grow. Bad habits

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Third in a series of editorials on Trends in Medicine.

in eating, lack of exercise, lazy reading, like all bad habits, are hard to eliminate. It is so obvious however that many of our most useful, productive, and creative citizens are hell-bent on destroying themselves by abuse or disuse of body and mind that the Aspen project can be genuinely classed as a venture in preventive medicine of the highest type.

The Aspen idea, conceived ten years ago by Walter Paepcke, comprises two parts—the Seminars or discussion groups which have been in operation for seven years—and the Health Program which began only in 1957. The marriage of the two under one roof—the Aspen Institute—was obviously logical and desirable, and was accomplished last summer. There is therefore one program which includes reading, discussion, planned exercise, various types of physiotherapy, and relaxation. Both the reading and the exercise are graduated, becoming more difficult as the days pass. The spirit is tended by periods of rest, and the beautiful surrounding scenery of mountains, forests, lakes and streams is naturally designed for contemplation and refreshment. Regularity in hours, various outdoor activities, the best in music, and adequate sleep round out the program.

The two-week course is an exciting experience which has converted even the most skeptical. It is difficult to describe without becoming fulsome. The entire program, conducted away from the stress and routine of one's daily life, is ideally suited to enable an individual to change his pattern. We believe that such a change, properly directed, may become permanent. Thus far about half of the "alumni" seem to have retained real benefit, and we hope have prolonged their lives and increased their productivity. The program is ideal for the patient mentioned in my opening paragraph, and it is the fundamentally well man who is however tending to destroy himself whom we are trying to save.

"Mens sana in corpora sano" could be our motto, if we were to have one. Instead, let us say that we are trying to prevent illness by developing better habits in mental and physical fields, and by increasing awareness of the inseparable relationship which exists between body, mind and spirit. This is the essence of the Aspen project, and it is this which makes it so bold and imaginative a venture in the field of preventive medicine.

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Aspen, Colorado

MEDICAL LIBRARIANS: WHAT DO THEY DO?

Sooner or later in the life of every medical librarian, some interested or curious person will ask, "*What* do you do all day with your time?" Or some one will stop at the door and say in a shocked voice, "Is this where you *sit* now?" Because the idea of medical librarianship as a career has not been as widely publicized as other fields of librarianship or as other professions associated with the care of sick in hospitals, the profession is evaluated negatively by the misinformed. To them a librarian appears to do nothing but sit at a desk keeping a custodial eye on the books and occasionally taking one off the shelf and putting it back again for a seldom-seen patron. Actually, the work is dynamic, often it is very exciting, and always, it is most satisfying, intellectually.

However, what a medical librarian does depends, first, on two factors: on her academic and personal qualifications and on what the institution which employs her expects her to do. If those who employ her do not realize that she has clearly defined functions within her own field, one of several conditions may exist. She may be selected on the basis of her ability to do "light typing," or she may be a former member of an active staff or faculty who is assigned to the library as the first plank to retirement. Or again, a person may be chosen to fill this position who has many extra-librarian aptitudes, that is, one who is readily convertible into a secretary, a public relations person or a handy man to manipulate the mechanical gadgets, the tape recorders, the slide projectors, and the movie machine. Unless a librarian is chosen with sound academic and personal qualifications, the type of work done in the medical library will be inferior and the library will make slow progress, if any.

Minimal academic qualifications for medical librarianship include a bachelor's degree with basic work preferably in languages, literature and the biological sciences. An additional year of study in an accredited library school is a requisite. Courses in medical terminology, bibliography and in medical librarianship are also essential preparation for the profession. The latter courses if taken in a university approved by the Medical Library Association prepares a librarian for Grade I Certification by the Association. Librarians may qualify for Grades II and III Certification by taking internships, again at approved universities, and by doing graduate work, both in librarianship and in medical subject fields. The purpose of certification is to raise the standards of the profession, to form criterion by which professional knowledge of the librarian may be judged and to give those who employ librarians a reliable guide for their choice.

As to personal qualifications the medical librarian must have those which make her adaptable to her specialized type of work. Medical librarians writing in their own literature single out essential traits. She must have a mind that prefers organization to disorder, a liking for books and for working with people, initiative, curiosity, imagination, a sense of humor, a strong constitution. Miss Mildred Crowe, librarian at the Alabama Medical Center says, "She should be a saint, a scholar, a teacher, and a technician."

The field of medical librarianship is a complex one. Much of what a librarian will do depends also on the size and type of the affiliating institution: agency, society, school or industry. While the basic principles of librarianship are the same in all types of special libraries, duties and services will vary according to the aims and objectives of the parent affiliation. The needs of clientele will be quite different in the National Library of Medicine, than they will be in a pharmaceutical research library, a medical society's library or in the library of a teaching or non-teaching hospital.

Other factors that affect the extent and the type of work a librarian does are: the size of the budget allocated to the library, the distance the library may be from larger and more comprehensive collections of the same subject matter and the needs or demands of its clientele, for in libraries as in all institutions with commodities, the user creates the demand.

The final word on the questions, "What do you do with your time?" depends, in great measure on the resourcefullness, imagination and judgment of the individual librarian. One person's remark, "I have never seen a medical librarian who is not happy in her work," is really the heart of the whole matter. Where work is done whole-heartedly in the spirit of dedication, there is practically no limit to its boundaries. This is more than true in the field of medical librarianship.

Sister Teresa Louise
Medical Librarian

LIFE INSURANCE IN ESTATE PLANNING TODAY

Is Your Wife the Beneficiary of Your Life Insurance Policies?

Under your present estate plan, you may have named your wife as beneficiary of life insurance proceeds which qualify for the marital deduction, while other assets are to go to your children. But assuming you are willing to give up your rights in the policy, it may now be advisable for estate tax purposes to reverse these dispositions by making the children the insurance beneficiaries and providing for the transfer of other assets to the wife. An irrevocable transfer of the policy to the children, or for their benefit, will remove the proceeds from your estate. And the full benefit of the marital deduction can be secured for other assets passing to the wife. If this is not done, all or part of the marital deduction may be wasted.

Do Life Insurance Policies Provide for Payments to Your Wife, with the Principal Going to Your Children or Others on Her Death?

If you die owning a policy with this settlement option, the entire proceeds will be fully includible in your estate with no reduction by reason of the marital deduction, unless your wife has the power to appropriate the principal, or at least to draw it into her estate. And even where provision of such a power does give the husband's estate the benefit of the marital deduction, any portion of the proceeds not used up by the wife during her life will be included in her estate at her death.

There is a way of satisfactorily providing for both wife and children and avoiding estate tax on the insurance proceeds in both estates—and this can be done without having to use the marital deduction. The insured transfers the policy to his wife irrevocably—taking care that neither the policy nor proceeds can come back to him or his estate. It is also provided that on the insured's death, the income from the proceeds is to be paid to the wife during her life. On her death the principal is to be paid to the insured's children.

Under this arrangement there is no estate tax on the proceeds when the insured dies, assuming the Treasury does not find that he transferred the policy "in contemplation of death." Nor is there any estate tax on the proceeds when the wife dies first—and thus dies owning the policy—the value of the policy will be included in her estate. Yet even this eventuality may be prevented by having a trustee hold the policy instead of the wife.

Furthermore, estate tax can be entirely avoided or reduced to a nominal sum even though the wife is given (in addition to income) the right to take part of principal during her life. This can be achieved by limiting her right to take principal to an ascertainable standard relating to her health, support, maintenance, etc. Such a right is classed as "nontaxable" and the unspent principal can go to the children free of any tax in the wife's estate.

Are Proceeds of Insurance on Your Life Payable to Your Wife Under the Interest Option?

Under the interest option, payments representing principal would not be taxable income to your wife, but payments representing interest would be fully taxable. Thus it may be to her benefit if the option is changed to give her the proceeds in installments.

That part of each installment which represents principal is tax-exempt. That part of each installment which represents interest (including excess interest dividends) is also exempt up to an amount not exceeding $1,000 a year.

There is no longer any income tax advantage in electing the installment option for beneficiaries other than the wife since any amount they receive in excess of the principal proceeds is fully taxable.

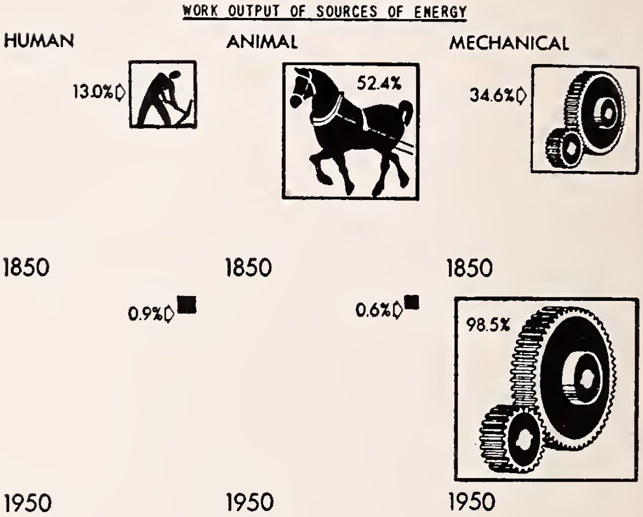
It may be helpful in your planning to know how much insurance payable in installments over the widow's lifetime would yield roughly $1,000 annually in interest so that only a negligible amount need be reported in gross income. There is no precise answer for any given case since it would be necessary to know the wife's age at the time payments begin—which can't be determined until the husband dies. But this can, of course, be estimated and the necessary amount of insurance approximated.

Assuming the husband's death when the wife is fifty-seven, her life expectancy would be twenty-four years. About $63,000 of life insurance would produce an annual payment of $3,636, of which $1,011 would be interest and $2,625 would be principal.

Alan W. Giles

ECONOMIC HISTORY

The Twentieth Century Fund, a non-profit research organization, has just published a new survey of the past and future potentials of the American economy. The current study is designed to present in simple language and graphic illustration some of the highlights of a previously published but highly technical treatise on this same subject.



A typical example of the many charts and illustrations in the study is the accompanying one which shows how mechanical power has almost completely supplanted animal and human muscle power since 1850.

This illustration is of particular significance because the study says that it is the increased use of power and machinery to do our work which has been largely responsible in our having the highest standard of living in the world. In this connection the study says:

> "Certainly high productivity is the key to the entire achievement. Rising productivity centers around a worker's ability to turn out an increasing amount of goods or services in an average hour's work. The plant, materials and management supplied by the employer, and the energy, skill and spirit of the worker are important in this process.
>
> "But neither the competence of management nor the efforts of the worker supply the most basic reason for the steep upward trend of American productivity. . . . The fabulous increase in our output per man-hour over the past century has been due to the steady expansion of our factories and productive plant and our skill in working out constantly better methods and processes and providing more and better machinery to back up the human worker with vast amounts of inanimate energy. . . .

> "There is the central principle of our success, the secret of our phenomenal rise in productivity. We shall encounter the results of this principle many times as we look into every part of our entire economic system to get the full aspect of America's new dimensions."

There have, of course, been many reasons advanced over the years as to why the United States should have found the unique combination to make us the richest nation in the world. We have little more than 6 per cent of the world's population and less than 7 per cent of its land area. Also, we have by no means a monopoly on natural resources. Perhaps, however, the following comments from the original Twentieth Century Fund study will bear reproduction as they offer some very compelling thoughts on this matter:

> "The relative 'classlessness' and fluidity of American society, the absence of an aristocratic attitude toward trade as an occupation, together with abundant opportunities for business success, created an atmosphere that attracted into business the gifted and superior youths who in the older countries might have chosen careers in the army, the law, or the ministry. In the United States, to a much greater extent than in other countries, business success was generously rewarded, not only in material terms but in the coin of social approval and public esteem.
>
> "These ideas and attitudes, developing within a 'hybrid population' that was spreading rapidly over a new continent, created a political and social climate favorable to invention and innovation, to the growth of individual enterprise, to the willingness to take risks and invest capital. America's unique industrial achievements are the result not only of favorable climate, soil and resources but to an indeterminable extent of these more subjective factors. A skilled, vigorous population—an individualistic, aggressive people with an extroverted approach toward themselves and their environment—has probably been more crucial to our technical development than the objective factors of climate and resources. In the last analysis, it is the attributes—and the attitudes and ideals—of the American people which account for the dynamic character of the American economic system."

From *Brevits*
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EDUCATION AND STATE GOVERNMENT

Education is not only one of the major responsibilities of state government, but it is also a function of government that is most essential to the progress and happiness, as well as the prosperity and security, of all the people.

Today there are new factors and trends in our society that contribute toward making our problems and responsibilities in the field of education more urgent and more important than ever before. Outstanding among these is the increase in our birth rate, that has placed an unprecented burden of expansion on our schools. The increased numbers of children, born in the early forties and later, have swelled the numbers in our elementary and secondary schools, and the oldest of them are just now about to enter college. Because of the low birth rate in the thirties, our college age population is now at its low point. But in the next few years the tremendous pressure of increasing numbers will reach our institutions of higher education.

Geographical changes in our population, primarily in the phenomenal growth of the suburbs, have made problems of school facilities there particularly acute. Social economic and technological changes in our society require an ever increasing proportion of people with higher education, not only in specialized scientific fields but also in the area of sound, broad, liberal education. And constantly increasing costs of education, together with the increasing cost of other governmental functions, make the problem of finance, and a wise sharing of these costs between local districts and the state, one that needs continuing study and evaluation.

I regard education as of such importance that I sent a special message to the 1957 Minnesota Legislature, making specific and detailed recommendations in this field. We have had the sincere, vigorous, and effective co-operation of the Legislature, of agencies and departments of state government, of school boards and of voluntary organizations of teachers, parents, and citizens. I am happy to report that I believe the progress made in legislation for education in Minnesota was greater at the last legislative session than at any other time in our history.

This progress was especially important in extending various kinds of special education, and in providing increased support both for elementary and secondary schools and for higher education.

We have stepped up our program in vocational rehabilitation so that in 1957 there were 967 rehabilitated as compared with 582 in 1955, and 679 in 1956. This increase is of even greater significance in view of the fact that the number of rehabilitations of persons classed as severely disabled was more than doubled—from 85 to 181—last year. This achievement, of course, required an increased staff; and we have adopted the policy of making grants to specialized agencies, such as the Rehabilitation Center at the University of Minnesota, to enable them to provide greater services.

Last year likewise marks a major step forward in educational services for exceptional children. A new law provides that every school district must provide special instruction and service for handicapped children of school age, and provides for financial and supervisory assistance for this program. Continued progress and improvement of the state program for children needing special educational will be assured by an act which created an Advisory Board on Handicapped, Gifted and Exceptional Children.

Public schools in Minnesota are supported by local school districts with substantial contributions from the state, plus comparatively small federal contributions, primarily in the field of vocational education. State aid in substantial amounts tends to equalize educational opportunity throughout the state. It also prevents the levying of a heavier tax burden on property—for the property tax is the chief source of revenue for local school districts. If the proportion of school support which comes from the state were increased, the tax load on property could be lightened, and we could carry out still further the principle of equalization. This would, however, mean still further increases in appropriations at the state level.

The people of Minnesota are giving increased attention to their schools. I am sure they recognize not only that education must be paid for, but also that it is something too valuable to be neglected.

Orville L. Freeman
Governor of Minnesota

GANGLIONIC AND ADRENERGIC BLOCKING AGENTS

(Continued from Page 251)

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FUNDAMENTALS OF ANTICOAGULANT THERAPY

(Continued from Page 255)

or peripheral arterial embolism, it is usually advisable to start treatment with heparin and a coumarin anticoagulant and to continue use of the coumarin agent as soon as its effect has reached the therapeutic range, the use of heparin then being discontinued. However, heparinization at the beginning of anticoagulant therapy usually is not employed after acute myocardial infarction.

Summary

The use of anticoagulants is a valuable therapeutic adjunct. It requires careful supervision, some experience on the part of the physician and close attention to the establishment and maintenance of adequate degrees of anticoagulant effect within the prescribed therapeutic range. For such supervision when one of the coumarin compounds is used, there is no substitute for the one-stage measurement of prothrombin time, which must be done at sufficiently frequent intervals to insure that an adequate but not excessive effect is being maintained. A small risk of bleeding is always present when anticoagulants are used, and this must be calculated in determining the advisability of such treatment for any individual patient. However, the threat to life by such bleeding is usually much less than the threat to life by the thrombosing disorder for which the treatment is considered.

President's Letter

A MOST REWARDING COMMITTEE—PHYSICIAN'S ASSISTANCE

To its members, the Physicians' Assistance Committee is the most satisfying committee of the State Medical Association. These men rarely miss a meeting, although they travel from the four corners of the State, and they return to their homes with interest and zeal renewed.

Visualize the situations that bring members of the committee together. The elderly doctor has for years taken care of the community. He has been its comforter in trouble, its first source of help in sickness, its aid in the presence of accident. He may have been an outstanding citizen, a pillar of the Church, a sparkplug for its youth program, the backer for school athletics, and always the guardian of the public health. He almost surely has helped many of the individual members of the community over hard spots, both physical and financial, and has often omitted sending his bills, or has actually spent his money to help the poor. As he grows old, he cannot work as hard, may have a stroke or break a leg, may even lose his memory, or his carefully planned investments may go sour. By this time, those old bills are uncollectable, but his expenses continue, and may increase.

The physician is proudly individual. He is reluctant to accept help from another individual, nor does he wish to be a public charge. He does not like the idea of a compulsory social security. Who, then, but his fellow physician can come to his aid? The Physicians' Assistance Committee was set up for just this function, assistance by his own profession to the physician in trouble.

The members of this committee have worked in a dedicated manner. The committee was appointed in 1953. Dr. E. R. Sather of Alexandria, Dr. E. A. Kilbride of Worthington, and Dr. Herman Juergens of Belle Plaine were Charter members and have not missed a single meeting in almost five years. Dr. James Blake of Hopkins, Dr. Paul Boman of Duluth, Dr. John Dordal of Sacred Heart and Dr. W. E. Macklin of Willmar have missed only one meeting in all that time. Such devotion is hard to find, and comes only from dedicated men who are convinced of the value of the cause. The project was the idea of Dr. W. R. Humphrey of Stillwater, who has been chairman until now. Dr. Humphrey himself has been forced to give up his activities because of ill health. His committee chairmanship has been taken over by Dr. James Blake who carries on with his usual thoroughness and enthusiasm.

It is, indeed, warranted that credit be given to these men who give so freely of their one completely irreplaceable resource—their time.

Horatio B Sweetser

President, Minnesota State
Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

MINNEAPOLIS TOPS U.S. ON MEDICAL CARE COSTS

Minneapolis is the highest priced city in matters for consumer costs for medical care. This disclosure was made recently in the monthly report of the Bureau of Labor Statistics.

For food, clothing, transportation and all other essential items combined, cost of living is higher in Boston, Chicago, Detroit, Portland and Los Angeles. But for medical care alone, Minneapolis' index of 171.6 is the nation's highest. Or at least it was at the end of January. This compares with the national average of 141.7, which represents a 0.6 per cent rise over the previous month.

The increase, said the Labor Department, "resulted from scattered reports of advances in fees for professional services and hospital rates. Group hospitalization insurance premiums also rose in a few cities."

RETAIL DRUGGISTS TESTIFY ON BEHALF OF JENKINS-KEOGH BILL

George H. Frates, Washington representative of the National Association of Retail Druggists in testimony given before the House Ways and Means Committee, urged passage of the Jenkins-Keogh bill to "correct a discrimination that has unfairly penalized those who work for themselves rather than for others." He declared:

> "Why should a corporation or an individual conducting some other form of a business who hires people, be permitted to defer a portion of taxable income from taxation by setting it up in a retirement fund, when the small, independent, retail pharmacist cannot do likewise? We have been told many, many times by the honorable members of Congress that small business is the backbone of the nation. If this be true, the Congress should overwhelmingly enact H.R. 9 and H.R. 10 into the law. These are days of challenge for small business—for the small self-employed person. . . . This proposed legislation would permit the individual operator to deduct from his total income a fixed percentage or dollar amount on which he would pay no income tax. He would be allowed to invest this money in a pension retirement fund. Only when he used this fund in later years would he pay income taxes on the money."

MEDICARE ISSUES RULES ON DRUGS AND ID CARDS

Medicare headquarters has continued authorizing of drugs to maternity patients by civilian physicians. ODMC Letter No. 9-58 makes this clear to dispel confusion or uncertainty in this regard rising out of earlier directives. To receive reimbursement for drugs supplied to maternity patients, either directly or through pharmacy channels, the physician should itemize the drugs and their cost on the claim form (DA Form 1863) or on an attachment thereto. Nomenclature, quantity and cost of the medicinals must be shown.

Many servicemen's dependents are presenting themselves for Medicare hospital and medical benefits without their official identification cards. ODMC Letter No. 10-58 points out that the doctor or hospital should call for one of the following, if the prospective patient cannot produce the regular card (DD Form 1173):

> Statement of the local commander that the patient is eligible dependent. Official documents or identification cards, other than DD 1173, attesting to eligibility for civilian services under Medicare.
>
> Statement by physician or hospital authority that he has personal knowledge of the patient, dependency status and eligibility for Medicare.

ODMC Letter No. 9-58 also deals with Medicare coverage of eligible dependents under age twenty-three who are in full-time study in institutions of higher learning. Such institutions must be listed in "Education Directory 1955-56, Part 3, Higher Education." This guide is purchasable ($0.50) from Supt. of Documents, Washington 25, D. C.

AGED LACK HEALTH INSURANCE

Only one-third of the nation's population over the age of 65 have any form of health insurance protection. This is one of the facts brought out as the result of a recent survey made by the New York State Joint Committee on Aging.

The survey points out that because of their high incidence of disease and disability, insurance companies are reluctant to underwrite medical expense for this age group, according to the Commission, and as a result, sickness too often brings medical indigency.

Currently, most companies will not provide coverage for the aged, and if they do, the premiums are considerably higher. One solution suggested has been a small premium increase for all age groups amounting to about 3 dollars per persons annually to absorb the additional cost of covering the aged; however commercial health insurance carriers maintain that competition will not permit increasing the premiums for younger policyholders, and many keep premium costs down by canceling individual policies at ages sixty-five, seventy or seventy-five. Others do not permit group policies, which comprise the largest proportion of health insurance, to be converted to individual contracts at retirement.

However, there are indications that the picture is growing brighter. Some private carriers are experimenting with covering for specialized groups of older people; for example, a company in Chicago recently wrote a group hospital-surgical contract covering more than 23,000 members of the National Association of Retired Employees.

The Health Insurance Institute reports 1957 saw expansion of health insurance to include protection for more and more of the population. Policies designed to afford coverage for retired workers and for those over sixty-five continued to increase in number. A recent nationwide survey indicated that the number of group insurance plans offering the retired worker the option to either continue his insurance under the group or to convert his health insurance coverage to an individual policy has more than doubled within the last four years alone. Recently introduced commercial health insurance plans with paid-up premiums at age sixty-five are also becoming increasingly popular, the Institute found, while significant advances were noted in the number of policies in force which guarantee renewal by the insurance company.

DISCIPLINARY ACT UPHELD BY COURT

The Supreme Court of Washington has affirmed the action of the disciplinary board operating under the Washington medical disciplinary act.

The board had suspended the license of a physician who was convicted of violating the internal revenue code by filing a false and fraudulent income tax return. This is believed to be the first case to have come before the Supreme Court of Washington under the medical disciplinary act which was adopted in 1955.

AMA NEWSPAPER TO MAKE DEBUT

The AMA News, a sixteen-page tabloid newspaper, will be introduced at the AMA annual convention in San Francisco, June 23-27.

The new publication will be distributed every two weeks to approximately 200,000 physicians.

Purpose of the new publication will be to bring to the attention of AMA members the multitide of projects and activities carried on by the association, as well as all non-scientific news of interest to the profession. Publishing a newspaper for members was selected as a means of improving communications between the association and its members.

Physicians will be kept informed of what is going on in medicine in all 48 states through *The AMA News.*

AMA GROWS

AMA membership of all classifications, as of September 30, 1957, rose to a record total of 168,399 members in all classifications. Included were 145,452 active members, 6,057 associate members, 16,512 service members, 289 affiliate members, and 89 honorary members.

NATIONAL JAYCEES HEALTH PROGRAM

More than 3,000 local chapters throughout the nation are participating in year-round health programs. Ten suggested activities have been planned including Salk Vaccine Campaign; Health Fair; Better Sight and Hearing for Children; Poison Control; Assistance to Mental Health for Youth; Adventures in Living for Older People; Help for Handicapped Children; Recreation for Mental Patients; Jaycee Health Forums; and Directory of Health Services.

U.S. MEDICAL POST IN DOUBT

A house bill introduced by Reps. Carl Vinson (D., Ga.), Leslie C. Arends (R., Ill.) and Paul Kilday (D., Texas), would kill the post of Assistant Secretary of Defense for Health and Medical Affairs. Dr. Frank B. Berry now holds the post created at the time of the Korean hostilities.

ARTHRITIS CAPSULES CURBED

A consent order restricting advertising claims for "Warsene Capsules" as a treatment for arthritis and rheumatism has been approved by the Federal Trades Commission. The order names a St. Louis firm and its former officers.

FDA CRACKS DOWN ON QUACKS

Six dubious therapeutic devices have been reported seized by the Food and Drug Administration in the month of January. Included were two taken from the office of a physician-chiropractor and used for cancer, congestion, etc.; another removed from a chiropractor's clinic because of peculiar labeling; and three seizures were made from a commercial firm whose representations were challenged by FDA.

UNITED STATES—RUSSIAN MEDICAL SCIENTIST EXCHANGE PLAN

A two-year period of multiple exchange programs of medical scientists has been agreed upon by the United States and Russia. Three or four medical scientists will be sent to the United States this year by the Russian Ministry of Health to deliver lectures and exchange experiences during a two or three week period. A similar group from this country will go to Russia.

BARBITURATE REFILLS QUESTIONED

Narcotics Commissioner, H. J. Anslinger, testifying before a House Appropriations sub-committee, has again questioned the advisability of issuing refills for barbiturate prescriptions. He told of one woman who was allowed to have a seconal prescription refilled 300 times "winding up, of course, in the insane hospital. . . . I do not think they should be allowed to be phoned in . . . that should not be." He said, however, that tranquilizers appear to have no attraction for narcotics addicts. "That is always a good sign, because when you see a drug addict interested in some new form of medication, some new synthetic drug, that is the one to watch. He is not interested in the tranquilizers."

FOURTEEN REASONS WHY THE FORAND BILL SHOULD NOT BE PASSED

The Medical Society of the State of Pennsylvania in the March *Newsletter* listed the following fourteen reasons why the Forand Bill should not be passed. They are:

1. It would destroy a significant area of voluntary health insurance coverage. The establishment of a compulsory federal system of providing hospital and surgical services for those eligible for social security benefits would terminate voluntary health insurance experimentation for the aged and result in the cancellation of a large number of policies. The aged would have no incentive to provide private health insurance coverage for themselves (approximately 50 per cent are now covered). Those now covered offer proof of the financial ability and willingness of a large segment of the aged population to acquire medical care without government interference.

2. Hospital and surgical services for the older population can be handled by community and voluntary means. In 1950, only 20 per cent of those over age sixty-five were covered by voluntary health insurance. Approximately 50 per cent now have some coverage. Assuming this expansion continues, within a few years virtually all of the insurable oldsters will be enrolled.

3. It would mean another assault on the pocketbook of the taxpayer. This plan would result by 1957 in an increase in federal OASI taxes of up to $213.00 annually ($106.50 for the employe and $106.50 for the employer.) This 59 per cent increase would result in annual payments of $285.00 for each by 1957. The tax for self-employed persons would be $427.50 annually. Proposed income tax reductions would be more than offset by these social security tax increases.

4. A large percentage of beneficiaries under the bill do not need assistance. All eligible social security retirement and survivor recipients, even though they are gainfully employed or able to pay for hospitalization and medical care, would receive benefits under the bill.

5. It would immediately nationalize a large segment of hospitalization and medical care. The federal government would control standards of hospital and surgical services and the conditions under which services are provided.

6. It limits free choice of hospitals and physicians. Only those hospitals and doctors entering into contracts with the federal government could participate in the program. Participation by hospitals and physicians could be arbitrarily restricted for administrative reasons.

7. The personal relationship between the doctor and his patient would be jeopardized. Government regulations would be imposed on patient and physician alike, bringing a third party into the relationship.

8. It would violate the basic concepts of the social security program. Since its inception, social security has provided benefits in terms of dollars which many people have used as a floor in planning their retirement needs. The interjection of the service benefit concept into the law (Which the Forand bill does for the first

time) would alter the basic program and would cause a financial drain that could wreck the retirement program. In fact, under the existing program, this year, for the first time, the system will receive less income than it will pay out in benefits.

9. It would stimulate over-utilization of hospital and medical services. In many countries where similar legislation is in effect, there has been a staggering increase in use of hospital facilities by those over sixty-five. (In Saskatchewan, Canada, the average person over sixty-five occupies a hospital bed 7.2 days a year; in the United States the average person over sixty-five occupies a hospital bed 2.5 days a year.) Over-utilization of hospitals would limit the number of beds available for the acutely ill of all ages in the community.

10. Community incentive to support voluntary hospitals would be virtually eliminated. Local communities would shift the responsibility for building and operating hospital facilities to the federal government.

11. The government would assume the role of price and wage fixer. Under the Forand proposal, the federal government would be given the right to fix the fees and charges to be paid to the participating physicians and hospitals. Historically, when such a principle as this is once established, it is extended to other segments of the population. The fixing of fees and charges by government would be a step toward a controlled and regimented economy.

12. Many aged persons would become unduly concerned with their health. By eliminating personal financial responsibility, many aged persons would seek unnecessary hospital and medical care for trivial and imaginary illnesses.

13. It would discourage family responsibility. Most families assist in caring for their aged members. The Forand bill would tend to destroy this traditional family responsibility and moral concept.

14. Other age groups would demand the same benefits. Soon the program would be extended to the entire population. This development would completely nationalize our hospitals and the practice of medicine.

SPICY SUGGESTIONS

Good news for patients on sodium-free diets—more than thirty different spices and other foods have been found to brighten the flat taste of salt-free foods and make them palatable. Suggested condiments include: Angostura Aromatic Bitters (used primarily in the preparation of alcoholic drinks), allspice, almond extract, basil, bay leaves, cardamon, carroway, chili powder, cinnamon, clove oil, cocoanut, curry, dill, garlic, ginger, honey, homemade horseradish, jams, lemon juice, lime juice, mace, marjoram, mushrooms, mustard seed, nutmeg, onion, paprika, parsley, freshly ground pepper, peppermint, pimiento, rosemary, saffron, savory, sesame, sugar, tarragon, thyme, vanilla and vinegar.—*American Journal of Nursing,* February, 1958.

LOCAL EDEMA OF LOWER EXTREMITIES

(Continued from Page 259)

Summary

The differentiation of edema with regard to local or systemic causes, the relative benignity of swelling of the lower extremity resulting from local causes and the reassurance of the patient that such edema is benign are important considerations. Proper and continuing treatment, especially of chronic venous insufficiency and lymphedema, although difficult, is necessary in order to prevent the complications and invalidism that are sequelae of improper treatment.

SURGICAL TREATMENT OF AORTIC INSUFFICIENCY

(Continued from Page 262)

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Minnesota State Medical Association

105th Annual Meeting

Minneapolis, Minnesota—May 22-24, 1958

BUSINESS SESSIONS

Hotel Leamington

TUESDAY, MAY 20

2:00 P.M.—Council
Dubonnet Room, Hotel Leamington

6:00 P.M.—Council Dinner

8:00 P.M.—Council
Dubonnet Room, Hotel Leamington

WEDNESDAY, MAY 21

8:30 A.M.—Council
Dubonnet Room, Hotel Leamington

10:00 A.M.—Reference Committees
Rooms to be assigned

2:00 P.M.—House of Delegates
Hall of States, Hotel Leamington

6:00 P.M.—New Members and County Officers Dinner
Town Hall, Hotel Leamington

8:00 P.M.—House of Delegates
Hall of States, Hotel Leamington

THURSDAY, MAY 22

8:00 A.M.—Committee Breakfasts
Rooms to be assigned

8:30 A.M.—Council
Dubonnet Room, Hotel Leamington

12:15 P.M.—House of Delegates
Hall of States, Hotel Leamington

FRIDAY, MAY 23

8:00 A.M.—Committee Breakfasts
Rooms to be assigned

8:30 A.M.—Council
Dubonnet Room, Hotel Leamington

SATURDAY, MAY 24

8:30 A.M.—Council
Dubonnet Room, Hotel Leamington

SOCIAL EVENTS

WEDNESDAY, MAY 21

Dinners

New Members and County Officers Dinner
6:00 p.m. Town Hall, Hotel Leamington

Golf Dinner
6:30 p.m. Edina Country Club

THURSDAY, MAY 22

Luncheon

Medical Women's Association
12:00 noon. Normandy Hotel, 405 South 8th St., Minneapolis. All women physicians are invited to attend the luncheon sponsored by Alpha Epsilon Iota Medical Sorority. Come and get acquainted and enjoy an interesting program. Dr. Harriet Blodgett, Director of Sheltering Arms School, will speak on "What Happens to the Retarded Child When He Leaves the Doctor's Office." Luncheon tickets are $2.50. Reservations must be made before May 15. Send checks to: Dr. Helen Dahl, 4708 Isabel Ave., Minneapolis, phone number PArkway 4-0179.

Dinners

Minnesota Academy of General Practice
6:00 p.m. Town Hall, Hotel Leamington. Tickets, $5.00, gratuity included. Speaker: Harry F. Dowling, Professor of Medicine and Department Head, University of Illinois College of Medicine, Chicago. His topic will be "The Doctor as a Teacher."

Minnesota Academy of Ophthalmology and Otolaryngology
6:00 p.m. Minnesota Club, St. Paul. Marshall M. Parks will speak on "The Importance of Early Strabismus Care."

Minnesota Radiological Society
7:00 p.m. Gold Room, Radisson Hotel. Tickets may be purchased at the dinner.

Minnesota State Medical Association
9:00 p.m. OPEN HOUSE, Hall of States, Hotel Leamington. All physicians and their wives are invited to attend the annual entertaining OPEN HOUSE variety program as guests of the Hennepin County Medical Society.

FRIDAY, MAY 23

Luncheon

12:15 p.m. *Minnesota Society of Clinical Pathologists.*

Dinners

Minnesota Society of Clinical Pathologists
6:00 p.m. Annual Dinner and Business Meeting, Curtis Hotel.

Annual Banquet, Minnesota State Medical Association
7:00 p.m. Hall of States, Hotel Leamington. Tickets are $5.00, gratuity included.

Speaker: David B. Allman, President, American Medical Association.

Introduction of Woman's Auxiliary President: Mrs. Reuben Erickson, Minneapolis.

Presentations:
Fifty Club Certificates
Southern Minnesota Medical Association Medal
Distinguished Service Medal

ROUND TABLES

Hotel Leamington—12:15 P.M.

THURSDAY, MAY 22

Office Gynecology..............James S. Hunter, Rochester
Hemorrhage in Obstetrics
Grant F. Hartnagel, Red Wing
Abuse of Steroids........Charles H. Slocumb, Rochester
How to Handle Drug Addiction
E. M. Hammes, Jr., St. Paul
Psychiatric Aspects of Obesity
John E. T. Haavik, Duluth
Causes of Gastro-Intestinal Hemorrhage
M. H. Stauffer, Rochester
The Sexual Deviate....Bernard C. Glueck, Minneapolis
Uses of Radioisotopes............................Elmer Paulson, Joseph Ryan, St. Paul

FRIDAY, MAY 23

Office Dermatology ....Robert R. Kierland, Rochester
Drug Therapy in Hypertension
James J. Coll, Duluth
Treatment of Post-operative Shock
Ellis N. Cohen, Minneapolis
Office Proctology..Charles A. Neumeister, Minneapolis
Fractures in the Upper Extremities of Children
D. L. McCain, St. Paul
Newer Insulins......................Moses Barron, Minneapolis
The Obstetrician's Role in Perinatal Mortality
John S. Gillam, Fargo, North Dakota

GENERAL SESSION

Minneapolis Auditorium

THURSDAY, MAY 22

Morning Session

Chairman: J. P. Medelman

A.M.

8:30 Visit Scientific and Technical Exhibits

9:00 EMERGENCIES—Panel Discussion—
Moderator, John E. Twomey, Minneapolis
Poisoning........Robert D. Semsch, Minneapoilis
Medical Cardiac Emergencies
Richard J. Frey, Minneapolis
Unconscious States..H. F. R. Plass, Minneapolis
Catastrophic Drug Reactions
L. E. Prickman, Rochester

10:15 INTERMISSION
Visit Scientific and Technical Exhibits

11:00 RUSSELL D. CARMAN MEMORIAL LECTURE
Radiation Hazards in Clinical Medicine
Richard H. Chamberlain, Professor of Radiology, University of Pennsylvania School of Medicine and Graduate School of Medicine, Philadelphia, Pennsylvania

Discussion
James Marvin, Ph.D., University of Minnesota
Donn G. Mosser, University of Minnesota

Afternoon Session

Chairman: Wm. D. Coventry

P.M.

12:15 Round Table Luncheons

1:30 Visit Scientific and Technical Exhibits

2:00 THE FAMILY DOCTOR'S RESPONSIBILITY TO HIS PR-SCHOOL EYE PATIENT
Moderator—W. J. Bushard, Minneapolis
Amblyopia ....Theodore Martens, Rochester
Visual Acuity..........Walter Fink, Minneapolis
Recognition of Strabismus
John McNeill, Saint Paul
Diagnosis and Management of Other Eye Problems
Marshall Parks, Clinical Instructor of Ophthalmology, Georgetown University, Washington, D. C.
Tracheotomy: When, Why and How
Robert E. Priest, Minneapolis

3:15 INTERMISSION
Visit Scientific and Technical Exhibits

3:15 Obstetric Manikin Demonstration
Grant F. Hartnagel, Red Wing

4:00 WHAT'S NEW IN DERMATOLOGY—Panel Discussion — Moderator, Francis Lynch, Saint Paul
Treatment of Superficial Mycoses
Harold G. Ravits, Saint Paul
Treatment of Acne
Frederick T. Becker, Duluth
Eczema of the Hands
Carl W. Laymon, Minneapolis
Use of Sedatives and Tranquilizers
Robert R. Kierland, Rochester

5:15 Visit Scientific and Technical Exhibits

6:00 Special Dinners

9:00 Open House....Hall of States, Hotel Leamington

GENERAL SESSION

Minneapolis Auditorium

FRIDAY, MAY 23

Morning Session

Chairman: J. E. Twomey

A.M.

8:30 Visit Scientific and Technical Exhibits

9:00 SURGERY—Panel Discussion
Micrococcus Infections in the Hospital—1958
Wesley W. Spink, Minneapolis
Fluid Balance and the Acutely Injured Patient
Bernard Zimmermann, Minneapolis
Management of Head Injuries
Wallace Ritchie, Saint Paul
Inguinal and Femoral Hernioplasty
Chester B. McVay, Clinical Professor of Surgery, University of South Dakota Medical School, Yankton, South Dakota

10:15 INTERMISSION
Visit Scientific and Technical Exhibits

11:00 MINNESOTA DIVISION, AMERICAN CANCER SOCIETY LECTURE

The Cell as a Mirror of Disease

James W. Reagan, Associate Professor of Pathology and Director, Laboratory of Cancer Research, Western Reserve University, Cleveland, Ohio

11:30 ARTHUR H. SANFORD LECTURE IN CLINICAL PATHOLOGY

The Spontaneous Regression of Cancer

William Boyd, Emeritus Professor of Pathology, University of Toronto, Toronto, Ontario

Afternoon Session

Chairman: R. N. Barr

P.M.

12:15 Round Table Luncheons

1:30 Visit Scientific and Technical Exhibits

2:00 VIRUSES AND VIRUS DISEASES—Panel Discussion

Moderator—Gaylord Anderson, University of Minnesota

Current Virus Disease Problems in Minnesota

Herman Kleinman, University of Minnesota

NORTHWESTERN PEDIATRIC SOCIETY LECTURE

Immunization With Orally Administered Attenuated Live-Virus Poliomyelitis Vaccine

Maurico M. da Silva, Communicable Diseases Branch, Pan American Sanitary Bureau, Washington, D. C.

Some International Aspects of Virus Diseases

Fred L. Soper, Director, Pan American Sanitary Bureau, Washington, D. C.

3:15 INTERMISSION

Visit Scientific and Technical Exhibits

3:15 Obstetric Manikin Demonstration

John S. Gillam, Clinical Instructor, Obstetrics and Gynecology, University of Minnesota, Fargo, North Dakota

4:00 WORLD HEALTH ORGANIZATION STUDIES AND REPORTS

Presiding Officer—Walter Judd, Washington, D. C.

Welcome to World Health Organization

Charles W. Mayo, Rochester

Address—Marcolino G. Candau, Director-General, World Health Organization, Geneva, Switzerland

Discussion—Leroy E. Burney, Surgeon General, U. S. Public Health Service, Department of Health, Education, and Welfare, Washington, D. C.

5:15 Visit Scientific and Technical Exhibits

6:00 Special Dinner and Meeting

7:00 Annual Banquet

Hall of States, Hotel Leamington

Presiding—O. L. N. Nelson, Minneapolis

Introduction of Woman's Auxiliary President: Mrs. Reuben Erickson, Minneapolis

Presentation: Fifty Club Certificates; Southern Minnesota Medical Association Medal; Distinguished Service Award

Address: H. B. Sweetser, President, Minnesota State Medical Association

Address: David B. Allman, President. American Medical Association, Atlantic City, New Jersey

GENERAL SESSION

Minneapolis Auditorium

SATURDAY, MAY 24

Morning Session

Chairman: W. S. Neff

A.M.

8:30 Visit Scientific and Technical Exhibits

9:00 MENTAL HEALTH—Panel Discussion—Moderator, William C. Menninger, Topeka, Kansas

The Thorny Youngster

H. S. Lippman, St. Paul

The Querulous Primigravida

Owen F. Robbins, Minneapolis

The Confused Senior Citizen

J. T. Pewters, Minneapolis

Physicians' Feelings

William C. Menninger, President, The Menninger Foundation, Topeka, Kansas

10:15 INTERMISSION

Visit Scientific and Technical Exhibits

11:00 MEDICO-LEGAL ASPECTS OF HEART DISEASE—Panel Discussion

Moderator—Judge Warren E. Burger, United States Court of Appeals, Washington, D. C.

Mr. Robert Faricy, Referee, Minnesota Industrial Commission, St. Paul

Harry E. Underleider, Director, Medical Research, Equitable Life Assurance Society, New York, N. Y.

Simon Dack, Assistant Clinical Professor of Medicine, New York Medical College, New York, N. Y.

R. W. Kissane, Professor of Medicine in Cardiology, Ohio State University, College of Medicine, Columbus, Ohio

Woman's Auxiliary

36th Annual Meeting

PROGRAM

THURSDAY, MAY 22, 1958

A.M.

8:30 Registration..Curtis Hotel

9:45 Executive Board Meeting and Luncheon.... The Woman's Club
Luncheon at 12:15 P.M. Tickets $2.50

P.M.

2:30 Hennepin County Historical Society Tour 2303 Third Avenue S.
Transportation provided. Cars leaving Curtis Hotel for Tour and Tea promptly at 2:15 P.M. and 2:45 P.M.

3:00 Tea......Minneapolis Art Institute, Fireside Room
All visiting women are cordially invited to be guests of the Woman's Auxiliary to the Hennepin County Medical Society

FRIDAY, MAY 23, 1958

A.M.

8:30 Registration..Curtis Hotel

9:30 Annual Meeting............East Room, Curtis Hotel
Open to *ALL* members of the Auxiliary

Presiding—Mrs. C. L. Oppegaard, Crookston, President

Pledge of Allegiance and Auxiliary Pledge—Mrs. F. W. Behmler, Morris

> "I pledge my loyalty and devotion to the Woman's Auxiliary to the American Medical Association. I will support its activities, protect its reputation and ever sustain its high ideals."

Address of Welcome—Mrs. Karl W. Anderson, Excelsior, President, Auxiliary to the Hennepin County Medical Society

Response—Mrs. D. E. Stewart, Crookston

Presentation of Convention Chairmen—Mrs. Conrad Holmberg and Mrs. Mancel Mitchell

In Memoriam Service—Mrs. Howard Satterlee, Lewiston

Reports

Election of Officers

P.M.

1:00 Annual Luncheon....Cardinal Room, Curtis Hotel
Tickets $2.50

Installation of Officers—Mrs. H. W. Quist, Minneapolis

Installation of President and Presentation of President's Pin—Mrs. L. P. Howell, Rochester

Presentation of Gavel

Greetings from the New President—Mrs. Reuben Erickson, Minneapolis

3:30 Post-Convention Board Meeting

7:00 Annual Banquet..............................Hall of States, Hotel Leamington

Presiding—O. L. N. Nelson, Minneapolis

Introduction of Woman's Auxiliary President: Mrs. Reuben Erickson, Minneapolis

Presentation: Fifty Club Certificates; Southern Minnesota Medical Association Medal; Distinguished Service Award

Address: David B. Allman, President, American Medical Association, Atlantic City, New Jersey

Committees

General Arrangements—Mrs. Conrad J. Holmberg and Mrs. Mancel T. Mitchell.

Reservations—Mrs. Harry B. Hall, Mrs. Roland F. Neumann, Jr.

Tickets—Mrs. James G. Myhre, Chairman; Mrs. D. Keith Millett, Mrs. Wm. E. Petersen, Mrs. O. J. Baggenstoss, Mrs. John W. Johnson, Mrs. John K. Grotting, Mrs. George H. Marking.

Registration—Mrs. Arthur C. Kerkhof, Chairman; Mrs. Harold F. Buchstein, Co-Chairman; Mrs. U. S. Anderson, Mrs. Henry W. Quist, Jr., Mrs. Robert E. Nord, Mrs. Harold E. Miller, Mrs. Dale H. Correa, Mrs. Howard A. Shaw, Mrs. D. M. Thysell, Mrs. Lloyd S. Nelson, Mrs. Nathaniel H. Lufkin, Mrs. Emerson E. Hoppes.

Hospitality—Mrs. Virgil J. Schwartz, Chairman; Mrs. James A. Johnson, Co-Chairman; Mrs. Lester W. Carlander, Jr., Mrs. Hewitt B. Hannah, Mrs. Walter K. Haven, Mrs. Sheldon M. Lagaard, Mrs. Sam Leonard, Mrs. Malvin J. Nydahl, Mrs. Peter J. Schultz, Mrs. Theodore S. Smith, Mrs. Thomas Wright, Mrs. Ferdinand A. Zinter.

Transportation and Tour—Mrs. Conrad I. Karleen, Chairman; Mrs. Elmer T. Ceder, Mrs. Clarence E. Arlander, Mrs. Harold T. Gustason, Mrs. Louis A. Fried.

Press—Mrs. Harold S. Ulvestad.

Flowers—Mrs. Robert G. Tinkham, Chairman; Mrs. Virgil Lundquist, Mrs. Ames W. Naslund.

Pages—Mrs. A. Cabot Wohlrabe, Chairman; Mrs. Willis M. Duryea, Jr., Mrs. William H. Card, Mrs. Kenath H. Sponsel, Mrs. Robert O. Quello.

Executive Board Meeting and Luncheon—Mrs. Robert E. Priest and Mrs. Edward T. Evans.

Tea—Mrs. Edgar A. Webb, Chairman; Mrs. John W. MacDonald, Mrs. Rolf M. Iverson, Mrs. Maynard C. Nelson, Mrs. Howard M. Frykman, Mrs. John P. Wendland.

Annual Meeting and Luncheon—Mrs. James F. Shandorf, Chairman; Mrs. Horatio B. Sweetser, Mrs. Earl T. Opstad.

Music—Mrs. Russell C. Lindgren.

History of Medicine in Minnesota

A MEDICAL HISTORY OF MEEKER COUNTY TO 1900

HAROLD E. WILMOT, M.D.

Litchfield, Minnesota

One hundred years ago the first permanent white settlers arrived in our county. The early history of Minnesota includes Meeker County as part of the Wisconsin Territory. This comprised an indefinite mass of land which was west of the Mississippi, south of the 49th parallel and on westward to the Missouri. Captain Zebulon Pike had assisted in the permanent arrangements for Fort Snelling, and the settlement of Pig's Eye was now overflowing to the steeper banks of the Mississippi on the north and west, to be known in the future as St. Paul. St. Anthony (Minneapolis) was a growing community, and the Indians were steadily being pushed westward.

The area for some sixty miles due west was known as the Big Woods, and to traverse it was an undertaking fraught with perils to quail most of the adventurous spirits of that time. However, the economic pressures in the eastern United States, plus the glowing accounts to induce immigration from Europe, plus the curative benefits of the climate, and the desire to "get rich quick" did produce the characters who became the original settlers of Meeker County.

Meeker County, due in large part to its geographic location, has always occupied a unique niche in the history of Minnesota and the Northwest. One of its first settlers was a doctor who won the dubious distinction of being the first white man to die within its boundaries and probably the first physician to die within the state of Minnesota. One of our pioneer physicians was elected president of M.M.A. in 1911 as one of the earlier "rural" presidents. The "Acton Massacre" which shed the first blood in the Indian uprising in 1862 occurred in our county. In later years the colossal Land O' Lakes started with the Litchfield creamery as its Number One unit under the guiding hand of John Brandt, one of our native sons.

Meeker County was chosen as the first political unit in the United States to be organized for bovine tuberculosis testing to eradicate the spread of tuberculosis to humans from this source. Again, our county was chosen as the first unit to conduct a free Mantoux test for all inhabitants. This, again, was number one in the United States. Apparently some of these things came about because our county can be roughly located as being one-third the distance from the southern boundary of the state in a northerly direction and one-third the distance from the eastern boundary westward. This gives it a central and rather cross-sectional location for an average Minnesota county.

The county is thirty miles from north to south and twenty-four miles from east to west. It includes seventeen and one-half congressional townships. It is roughly 1,200 feet above sea level and consists of sandy loam of remarkable fertility. The eastern, western, northern, and southern portions are wooded and rolling with smaller hills, but the central portion is a flat sandy area, the first indication west of the Mississippi of the great prairies to follow if one journeys westward.

At present diversified farming is basically the economy, but in the early years fuel wood, railroad ties, and wheat were paramount in supporting its people.

Meeker County was created by the seventh territorial legislature of Minnesota, February 23, 1856. Some changes were later made in its boundary by annexing land from Renville County and relinquishing land to Kandiyohi County, but since 1869 the boundaries have become permanent.

At present the population is some twenty thousand people, with Litchfield the county seat and centrally located. Our name derives from the Honorable B. B. Meeker, a judge of the courts in Minneapolis.

In the words of Hon. A. C. Smith: "Why or wherefore is unknown, unless indeed, the Italian (sic) climate of the 'big prairie,' the richness of her soil, and the beauty of the surroundings were found typified in the geniality of the judge's temperament and the quiet good natured rotundity of his person."

According to the historians, Meeker County was probably not penetrated by La Verendrye, Hennepin, Radisson and others, though doubtless the products of this area were seen and appreciated by the courts of France. On government maps accompanying the official report of J. N. Nicollet and J. C. Fremont in their surveys of the hydrographic basin of the Mississippi in 1842, we find Meeker County to have been a part of the reservation for different sub-Indian bands. This was termed M-de-wa-kan-ton country. Nicollet recognized it as possessing the most beautiful lakes in Minnesota, and hence the name Kandiyohi was given to our western neighbor county, which is the Indian term for beautiful lakes, and originally included in our area. Early French maps alluded to this area as the "Coteau du greene Bois," a plateau heavily wooded and the height of land between the Minnesota and Mississippi drainage basins.

Access would only have been by canoe through the ramifications of the Crow River. This mild-flowing stream divides into two branches, the north and the south, just east of our boundaries; and the north branch originates in Stearns County to our northwest, while the south branch starts in Kandiyohi County to our west. Beaver and other fur-bearing animals were very prevalent in this area in the early days, and the Sioux understandingly were exceedingly reluctant to relinquish these hunting grounds to the "pale faces."

From St. Paul on westward the "Big Woods," as it was called, extended all the way to Meeker County. This was a huge hardwood forest, broken only intermittently by meadows and channels of small streams. However, at Meeker County the terrain changed and prairies took over in the central and western portions of our boundaries. This had much effect on the early colonization, for all one had to do was turn over the soil and plant. Eastern reaches, however, required much grubbing and land clearing before a crop could be raised. This wood was essential to build cabins and still can be found on eminences thoroughout the county.

Apparently the first permanent settlers arrived in September, 1855. Interestingly enough, they came in search of pine woods to aid in supplying lumber for the rapidly expanding needs of our young country. John W. Huy and Ben Brown poled a canoe up the Crow (Hasson) River as far as the present site of Forest City, which was later to become the county seat. They erected a cabin, staked claims and a town site, and remained. The first farming operation was performed by Benjamin Dorman when he "broke sod" and planted in 1856 on his farm east of Forest City.

In the autumn of the same year **Dr. Frederick Noah Ripley** arrived overland from Shakopee via Glencoe in company with John McClelland and established a temporary camp at Cedar Lake (Red Cedar Island Lake in Indian lore). They filed claims here and expected to remain, contingent upon the arrival of supplies by wagon in the early spring.

Previous to their arrival in Meeker County, the doctor had resided in Shakopee at the "Warren House" from September, 1853. He had been educated in New York City and had left his mother and fianceé there when he started his pioneer trip. In 1855 he had so favorably impressed the territorial authorities in St. Paul with his "refined and gentlemanly appearance" that he was notified, pursuant to his filing a claim in Meeker County that year, that he would be appointed a county commissioner in the newly organized county of Meeker. He was a "regular" doctor and anticipated practicing medicine in the usual manner in the growing community of Meeker County and expected to be married as soon as he could establish his house. His fianceé later became a school teacher in Glencoe.

On March 1, 1856, he and McClelland undertook a journey to the home of John Huy and Ben Brown. This, of course, was on foot and covered about eighteen miles each way, and was made because their supplies were low. They had been living in a rough "lean-to" shelter with the knowledge that ample supplies were always available in the cabin of Mr. Huy and Mr. Brown as arranged previously. Let us follow the words of an early historian, Attorney A. C. Smith, in *A Random Historical Sketch of Meeker County,* pp. 114-117:

> "It was a delightful morning—the sun shone brightly and the snow was melting fast till 9 a.m. when a gentle breeze from the north-west started up—in a short time clouds began to appear and by 12 a full-grown BLIZZARD was upon them.
>
> "They traveled as near a north-west course as they could calculate, until dark when they stopped in a small popple grove; gathering a pile of dry sticks, with which they started a fire, they camped for the night. . . . Imagination will tell how they spent the night, without food or blankets, and the thermometer down to 20 below and the wind blowing a hurricane, their only occupation was to hunt wood and keep up the fire till morning when they again started out, as was supposed in a direct course for Forest City and traveled until about 9 o'clock a.m. through snow from one to three feet deep before striking Crow River, but whether above or below Forest City they could not tell, and after a search for several hours both up and down stream the Doctor became discouraged and both started to go back whence they came—this was about two p.m.—by this time the storm had abated and the sun came out just before night.
>
> "About dark they accidentally came to the place where they had camped the night before, but to their great disappointment the fire had gone out and their few remaining matches were found to be wet, they had no fire and as they started out without food, in anticipation of getting through to Forest City the same night, hunger and cold told on them terribly. The Dr. was badly chilled—both walked the grove for some time but concluded to strike out for camp as it was quite as easy to walk on the prairie as in the timber.
>
> "Ripley showed signs of fatigue and quite frequently wanted to stop and rest—McClelland insisted it would not do to stop as he would freeze and McClelland was already aware that his own feet were partially frozen.
>
> "Ripley was evidently freezing as at every step he seemed to get weaker and less able to proceed and finally fell down in the snow—McClelland helped him up and led him on for some distance, until Ripley said "Mc. go ahead and if the teams have come into camp—have them come after me.'
>
> "McClelland very reluctantly left Ripley and made as rapid steps for camp as possible, knowing full well that he was taking a last farewell of the good man on earth.
>
> "McClelland left Ripley about a mile from what is now called Lake Ripley.
>
> "The Dr. seems to have wandered back to the grove, where his remains were found in the April following by Mr. William S. Chapman (now of California.)
>
> "McClelland left Ripley about 8 o'clock a.m. of the third day out and seven miles from camp. McClelland had a hard day's work and got into camp about sun down with his feet badly frozen, where he lay nineteen days before the expected team arrived. Dr. Ripley was thirty-two years old.
>
> "When McClelland was discovered in camp, he was in a dreadful state of prostration, was immediately removed to Shakopee and both legs amputated above the knee."

McClelland surmised that Dr. Ripley's remains were devoured by wolves as he himself had experienced difficulty in keeping the wolves off while in the cabin.

A few notes taken from the local *News Ledger* are instructive:

June 26, 1873: "The body of Dr. Ripley who froze to death on the prairie between Round Lake and Lake Ripley is to be disinterred soon and buried in Ripley cemetery."

June 28, 1878: "Burial services Sunday, June 16th. Reburied Dr. Ripley by Masonic Lodge. Burial Services: Frederick Noah Ripley, The first white man who died in Meeker County.

"On Monday last a committee appointed by the Masonic order of this place reported to the spot where Dr. Ripley was lain 22 years ago last April and exhumed the remains for a more fitting internment in a lot which had been set aside by the cemetery association for that purpose. Dr. Ripley's remains were found and buried by William S. Chapman in April, 1856. His grave was beneath an oak tree a short distance southeast of the residence of Ole Johnson and tradition informs us that his frozen corpse was found in a half-sitting posture at the foot of the tree under which he was buried. The remains were found in a better preserved conditon than was expected, the bones being all in a perfect state of preservation. A fitting casket was procured by the lodge and the remains were carried to the Masonic Hall to await the burial ceremony on Sunday last.

"At two o'clock the Masons assembled and formed in due and ancient style and headed by the Litchfield brass band and followed by quite a procession of citizens, escorted all that was left of Dr. Ripley to the cemetery. At the grave the usual Masonic services were held: the white apron and sprig were dropped upon the remains, and Dr. Ripley now sleeps in an honored grave on the banks of the beautiful lake that perpetuates his name.

"The following very appropriate remarks were made by P.W.M. Judge A. C. Smith as the remains were consigned to their last resting place:

" 'My brethren! The occasion which calls us together is of no ordinary character.

" 'Among the many incidents pertaining to the settlement of a new country, there are none which appeal to the better sympathies of humanity than those which apply to the hardships of the ones who first take possession of the new Eldorado.

" 'We today deposit in the central rotunda of the Litchfield cemetery the remains of brother Frederick Noah Ripley.

" 'Of the history of Brother Ripley, it is unnecessary for me to speak; it has passed upon the record and become a portion of the permanent history of Meeker County. The name attached to yonder beautiful lake will be thus preserved through coming time.

" 'Twenty-two years ago, the frail tenement, represented today by the remains before us, gave up a valuable life on this then boundless and unsettled prairie; pillowed upon the snowbank—lost to the world—thousands of miles from relations, and hundreds of miles from friends. God only knows the sufferings of a man in like circumstances; and why it was that the talented and accomplished Ripley should be required to lay down his life, on this spot, in the bloom of early manhood, must forever remain a sealed book to mortal eyes.

" 'Suffice to say, Brother Ripley fell in with the then current of immigration and, while he was among the first to interest himself in the settlement of central Minnesota, he was the first to surrender life on this vast prairie.

" 'He died on the third day of March, 1856; and about the middle of April following, his remains were discovered by William S. Chapman, then of Minneapolis, now of California, who came to Forest City, and with the assistance of James Quick, Dudley Taylor, Matt Standish, and Rudolph Schultz, a plain coffin was provided from the scant material then to be had and a decent burial was given him in yonder grave. That narrow and somewhat homely tenement he has occupied for twenty-two years.

" 'In the meantime our county is covered over with the civilization of the age; Meeker and Kandiyohi counties now shelter more than twenty thousand inhabitants, from all parts of the civilized world. Our fields teem with the richest gifts of Ceres; luxurious dwellings now adorn our hillsides and peep from numerous groves surrounding beautiful lakes, and the stranger is entranced as he silently gazes on the scene spread out before him. Railroads pass us, over five hundred miles and we are in daily communication with all civilized countries of the globe.

" 'A town (Litchfield) has sprung up but a single mile from the cheerless death bed of Ripley, now containing twelve hundred inhabitants with five church edifices pointing to the sky, and with a Masonic Lodge of over seventy-five members, and it well becomes us, although at this late day, to pay this tribute to the memory of our deceased brother.

" 'My brethren! The obligations of Masonry are never circumscribed by the statute of limitations and should the kindred of our brother ever learn of this deep doings, it may be a consolation to them to know that the emblems of innocence and immortality have been strewn with a liberal hand over the final resting place of our brother by Golden Fleece Lodge Number 89, A.:F.: and A.:M.:

> " 'My brethren, we have searched for and found the remains of our deceased brother, and here deposit them in the Temple of the Dead in form and with the ancient solemnities of our order, ever bearing in mind that the little line that divides the living from the dead is narrow and brittle and on either side lie the tragedies of human life and all of the mysteries of the future.' " . . . A. C. Smith, P.W.M.

Perhaps it would be well at this time, namely 1873, to glance at the county again. The lands had been largely settled throughout its reaches.

The largest village was the county seat of Litchfield in the center of the county. Eden Valley was just being established astraddle the county line in the north central boundary. Darwin was a growing village seven miles east of Litchfield, and Dassel well established twelve miles east of Litchfield. Cedar Mills was a thriving hamlet eleven miles to the south of Litchfield, and Swede Grove or Grove City, eight miles to the west. There were post offices in Kingston in the northeast corner of the county, Forest City (the original county seat) seven miles northeast of Litchfield, Manannah thirteen miles northwest of Litchfield, Crow River five miles west of Manannah, Rosendale nine miles south of Grove City, Cosmos southwest of Grove City sixteen miles, Greenleaf eight miles south of Litchfield, Cedar Mills four miles south and west of Greenleaf, Bonniwell Mills, in the extreme southeast part of the county, and Collinwood four miles southeast of Dassel. These inland towns, having no railroad, had stage coach service regularly from one to six days per week.

Immigration had been principally from native-born "Yankees" of the eastern seaboard states during the fifties and sixties, but an abrupt change had begun by 1870. Irish settlers had developed a community at Rice Lake (now Darwin) and had communicated with their friends and relatives, who in turn arrived in ever larger numbers. Germans had found the townships of Greenleaf and Ellsworth much to their liking, and the same process had taken place there. Norwegians and Swedes came and settled throughout the county but with perhaps the greatest numbers in the central and western portions. Indeed, Grove City was originally called "Swede Grove" on that account, and a township still carries that well-deserved appellation. Later still a large group of Finnish people established their homes in the northeastern corner of the county, principally in Kingston township.

Consequently, we are a well-mixed cosmopolitan race and justly proud of it. Intermarriage has naturally taken place, and minor groups and individuals including French, Indians, Bulgarians, Mexicans, Jewish, and Armenians, to mention a few specifically, have lived in and enjoyed the benefits of Meeker County citizenship. Perhaps the most interesting thing about the settlement of our county can be stated in this manner: Anyone coming here in pioneer fashion expected to have to work hard and build his own future with his own hands. His future and that of his progeny was assured if he was willing to combat the elements and take advantage of the productivity that nature provided. Of necessity he had to be strong physically, self-reliant, careful and frugal. These characteristics are typical of any Minnesotan today.

These pioneers of many extractions took their place well in the Civil War. The G.A.R. (Grand Army of the Republic) was well organized in Litchfield and in 1883 built and consigned their building to the city of Litchfield. Again, we must state that this was another first for Meeker County, inasmuch as this was the only meeting place owned by the G.A.R. in the entire state of Minnesota. At the outbreak of the Rebellion of 1861 there were twelve hundred inhabitants in Meeker County, and in summation we find two hundred forty-nine men who were regularly inducted as soldiers from this county. This probably is also a record.

Naturally, one looks for an explanation of such a situation, and it is readily available. The bloody Indian uprising of 1862 started in Meeker County. The Indians under Little Crow had been harassed and brow-beaten, always shunted farther westward, and Meeker County was at that time a frontier of farmers that extended into the Indian country. Let us again follow an early historian, A. C. Smith, in *A Random Historical Sketch of Meeker County*, p. 38 ff.

> "The Indian War formed a tragical episode in the history of Meeker County.
>
> "About 11 o'clock a.m., of Sunday, the 17th of August, 1862, the first deliberate massacre of the brutal Sioux outbreak, took place thirteen miles west of Forest City in the town of Acton.
>
> "Six Indians came first to the house of Robinson Jones, and thence to that of Mr. Howard Baker, where they deliberately shot five persons, viz: Robinson Jones, Howard Baker, Mrs. Ann Baker, Viranus Webster and Miss Clara D. Wilson, (the last named, at Jones' house). This was the commencement of that terrible Indian scourge, which resulted in the massacre of about nine hundred whites, on the frontier of Minnesota in the fall of 1862.
>
> "The preliminary tragedy at Acton, was not the result of a drunken riot—but was the commencement of a premeditated design to exterminate the whites from this region of country, although probably this early commencement, by an insignificant band, was not a part of the programme of Little Crow and other leaders. I allude to this, to correct errors which appear to have already been manufactured into departmental history. The Secretary of War reported to Congress, and all the pretended histories yet written, CRAW FISH to the fur traders, and allege a drunken broil as the commencement of the affair, and also make statements credited to the reports of a child afterwards found in Jones' house on the eve of the 17th, about 8 o'clock. Mr. John Blackwell, a reliable citizen, now deceased, found a little grandchild of Mrs. Ann Baker, eighteen months old, on the floor in Jones' house (the only one left or found in the house) and took it away—this child was too young to talk and was totally unconscious of its tragic surroundings.
>
> "It was lying upon the floor where it appeared to have cried itself to sleep.
>
> "Whether the Indians considered the child too insignificant to kill, or did not see it at all, cannot be known, the latter supposition is probably correct.
>
> "One writer says that the child lay on the bed and witnessed the scalping of his sister, but this is a mistake. The bed had not been tumbled and no other act done indicating that the Indians ever went into the house, and the girl had not been scalped or mutilated in any way but lay partly upon her back in a pool of blood just where she fell.
>
> "After Baker and Webster ceased to breathe, their wives started for the house of Mr. John Blackwell, their nearest neighbor, Mrs. Baker carrying in her arms an infant child preserved from the massacre.
>
> "When they reached Blackwell's they found no one at home, and proceeded on to the next neighbor, named Olson, a blacksmith, whom the Indians afterwards killed.
>
> "Late in the afternoon Blackwell, on horseback, came riding leisurely home, and learned from Ole H. Ness, Esq., whom he met on the prairie, the terrible news, which he at first could not believe, but Mr. Ness advised him to go to the house of Olson where the women then were and learn the particulars from them which he did, and learned from them that their husbands were dead before they left the house, and that two other persons, Robinson Jones and Mrs. Baker (mother of Howard Baker) were both shot, were both in great agony and evidently dying; that Mrs. Baker was lying in the house and Jones in the yard near the house; that the latter came there from his own house but a short time before where he had left the said niece and child.
>
> "The fate of those children was then problematical, fearing the worst, the Indians having gone in that direction, Blackwell concluded at once, that to find out what had become of them was an imperative duty, and immediately rode back to where he had left Ole H. Ness and found him with Henry Halverson, A. Nelson Fosen, and several others who had assembled and were discussing the matter.
>
> "The men were all in favor of going at once to the scene of the tragedy and securing, if alive, the girl and child.
>
> "It was after dark when they arrived at Jones house and the child was found alive—the remainder of the story has been told, and needs not to be repeated.

"The child was brought to Forest City and kept some months by Mr. and Mrs. Jewett and subsequently placed in charge of Mr. Charles H. Ellis of Otsego, Wright county, since which time we have lost track of him.

"Jones gave the Indians no liquor, and while there was liquor in Jones' house, up to the time of the inquest on Monday afternoon, there was no appearance of its having been molested. At the time of the inquest all the liquor in the house was poured on the ground.

"To show the evident design of these Indians to commit the tragedy at this point, we give the testimony of the wife of Mr. Howard Baker at the coroner's inquest conducted by A. C. Smith, the Judge of Probate and acting County Attorney. Her testimony was as follows:

" 'About 11 o'clock a.m. four Indians came into our house, staid about fifteen minutes, got up and looked out, had the men take down their guns and shoot them off at a mark, then bantered for a trade with Jones. About 12 o'clock two more Indians came and got some water; our guns were not reloaded; the Indians loaded their guns in the dooryard; I went back into the house, did not suspect anything at the time; supposed they were going away; next I knew I heard the report of a gun and saw Webster fall; he stood and fell near the door; another Indian came to the door and aimed at Howard Baker and shot; did not kill him at that time; he shot the other barrel of his gun at Howard and he fell.

" 'My mother walked to the door and another Indian shot her; she turned to run and fell into the buttery; they shot at her twice as she fell. I tried to get out of the window, but fell down cellar; saw Mrs. Webster pulling her husband into the house, don't know where she was prior to this; Indians immediately left the house; while I was in the cellar I heard firing out of doors.

" 'Jones said they were Sioux Indians and that he was well acquainted with them. Two of the Indians had on white men's coats; one quite tall, one quite small, one thick and chubby and all middle aged Indians, one had two feathers in his cap and one had three. Jones said "they asked me for whisky but I would not give them any".'

"This testimony shows a deliberate intention to massacre Jones' family. The facts are that Robinson Jones kept a sort of frontier public house and kept various articles of groceries, etc., with which he used to traffic with the Indians, with whom he was well acquainted, and obtained their furs and other proceeds of their hunting expeditions, and they had by some means got into his debt 40 or 50 dollars, which sum Jones had made arrangements to have paid out of their annuities.

"Certain Indian traders claimed the monopoly of the fur trade, and had for some years been in the habit of making advances to the Indians with the understanding that the Indians were to return to the traders the proceeds of the chase—the balance if any to be jerked (in a manner only known to Indian traders) out of the next succeeding annuities.

"Jones' little traffic was interfering quite materially with those traders, and was setting a bad precedent, and this may, perhaps, furnish a better clue than whisky, to the destruction of Jones' family, and which in its results, produced far more than the traders bargained for. The Indians were dissatisfied with all the traders, and Jones with the rest.

"Any one who understands the Indian trading system, as sanctioned by the Indian Department at Washington, can fill up the balance of the picture—those who do not, will never know any more about the origin of the Indian massacre than they do now.

"On Monday morning the 18th of August, news of the Acton massacre reached Forest City, and in less than an hour A. C. Smith, J. B. Atkinson, Milton Gorton and a few other accompanied by Mrs. Gorton and Mrs. Jewett, were on their way to the scene of the tragedy—with increasing numbers they arrived at Acton some sixty strong—held an inquest and buried the dead. On their return in the evening, the whole community was in a panic and appeared to be in Forest City.

"There were various men among our people, who 'lived fast,' between the 17th of August and the 4th of September, 1862—some in tragedy and some in comedy—far more peril to individuals than to companies of men, assembled for mutual protection.

"We could wish that we had a sketch of all such, including the hair-breadth escapes of men and families; but the facts are not furnished us, and we cannot do justice to the parties in interest from rumors obtained at the time, or from our recollection of circumstances." (pp. 58-59, *History of Meeker County*).

"On the 24th of August, Capt. Strout was ordered to Forest City via Glencoe and Hutchinson but deeming Forest City the safest place, from his stand point, came up the Mississippi direct to the latter place, arriving on the eve. of the 27th, and went into camp near the law office of Judge Smith." (p. 64, *History of Meeker County*).

"There were twelve persons killed in Meeker County and twelve in Monongalia the names of which were, in Meeker, Robinson Jones, Ann Baker, Viranus Webster, Clara D. Wilson, Philip Deck, Joseph Page, Linus Howe, Wilmot Maybee, Nels Olsen, Caleb Sanborn and Cross, and in July, 1863, James McGannon. In Monongalia three Olsen's, father and two sons, Anderson and son, Carl Carlson and son, Mr. Backland, Mr. Lawrenson and the Lumberg family, and nine in McLeod County, contiguous to Meeker, viz: Mr. Spondy, wife and two children, one child of John Adams (taking John Adams prisoner) and four of the White family at Lake Addie. Mr. Adams was taken prisoner Sept. 4.

"Total killed in Meeker and vicinity, thirty-three, and probably some who have never been reported.

July 1, 1863, McGannon was shot between Kingston and Fair Haven, probably by Little Crow in person, as this distinguished chieftain was a few days after shot by Mr. Lamson on section 30 Town 118-29 (Collinwood) Meeker County, and was found in possession of McGannon's coat.

"The great native warrior, together with his son—young Crow—were quietly making a dinner of raspberries, when the Irishman's bullet called him to his final account." (pp. 82-83, *History of Meeker County*).

This then gives us a background in the struggle for a pioneer's livelihood. Let us visualize a few of the other obstacles.

In 1873 we have the story of a natural catastrophe which is best described in the words of a paragraph in the *Minneapolis Journal* of January 7, 1953:

"TODAY MARKS ANNIVERSARY OF '73 BLIZZARD. Today is the anniversary of the great blizzard of 1873. It was known for sustained fury. For fifty-two solid hours an unrelenting wind blew, piling up huge drifts and cutting visibility to zero.

"The blizzard was especially severe in the Red River valley and southwestern Minnesota, where three days of storming were reported. It followed a succession of steady snowfalls and erupted after calm weather had given many farmers opportunity to get to town. Many of them were caught by the storm.

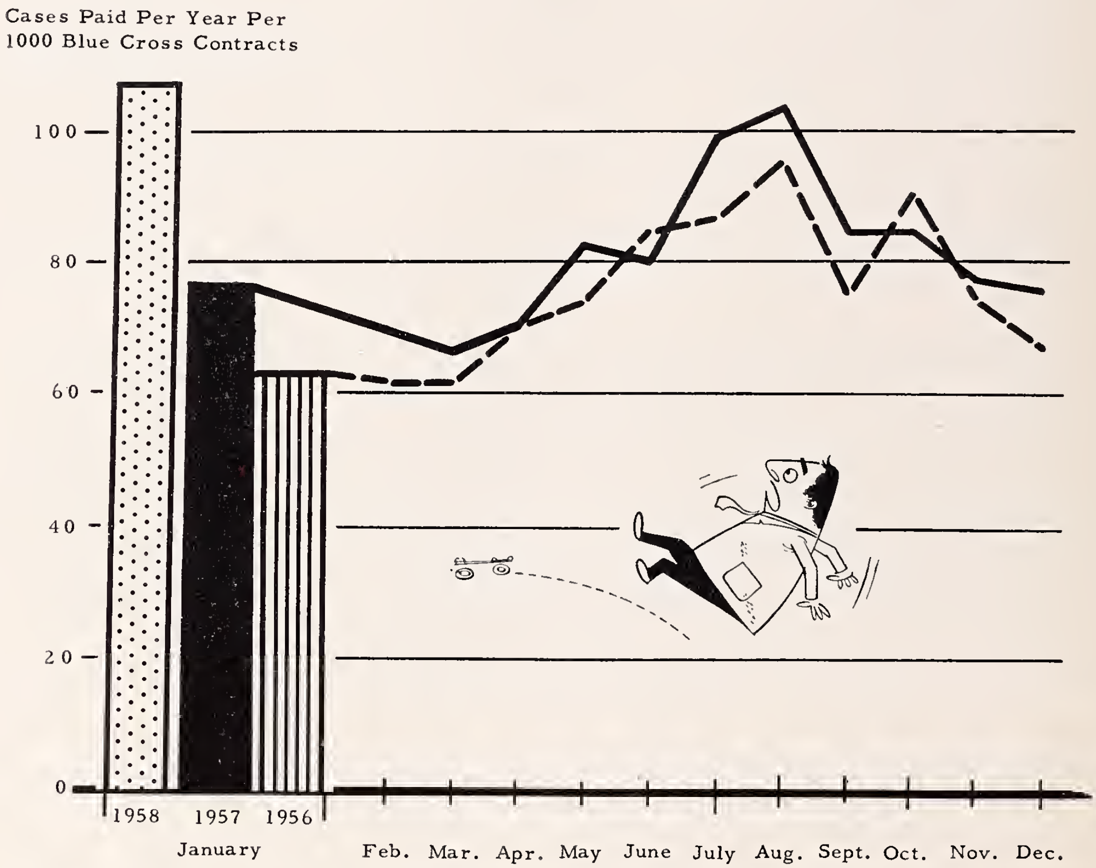
"Seventy persons died in the state. In one case a boy who started from school for his home across the road was found frozen to death eight miles away. The summer of 1873 wasn't so good either. It saw the state's first great infestation of locusts."

(To be continued in July issue)

MINNESOTA BLUE CROSS-BLUE SHIELD

Minnesota Blue Shield has experienced greater increases in the number of Medical Service Reports submitted during 1957 and the first two months of 1958 than during any similar period in the Plan's history.

TREND OF ACCIDENTS INCURRED BY BLUE CROSS SUBSCRIBERS



During 1956, Blue Shield received 212,828 of these claim forms, for a monthly average of approximately 17,700 while during 1957 more than 260,000 reports were received for a monthly average of over 21,600 claims. This monthly average figure is more than 22 per cent greater than the monthly average for the year 1956. During January and February of 1958, over 48,000 of these reports were received, an average of 24,000 reports each month. This figure is approximately 11 per cent greater than the monthly average number of reports received during 1957.

Blue Shield enrollment experienced an increase of approximately six per cent in the period between December 31, 1956, and March 1, 1958. However, increased enrollment alone does not account for the increased number of Medical Service Reports submitted. The increased number of claims submitted is principally due to increased utilization of the contract and increased incidence of services.

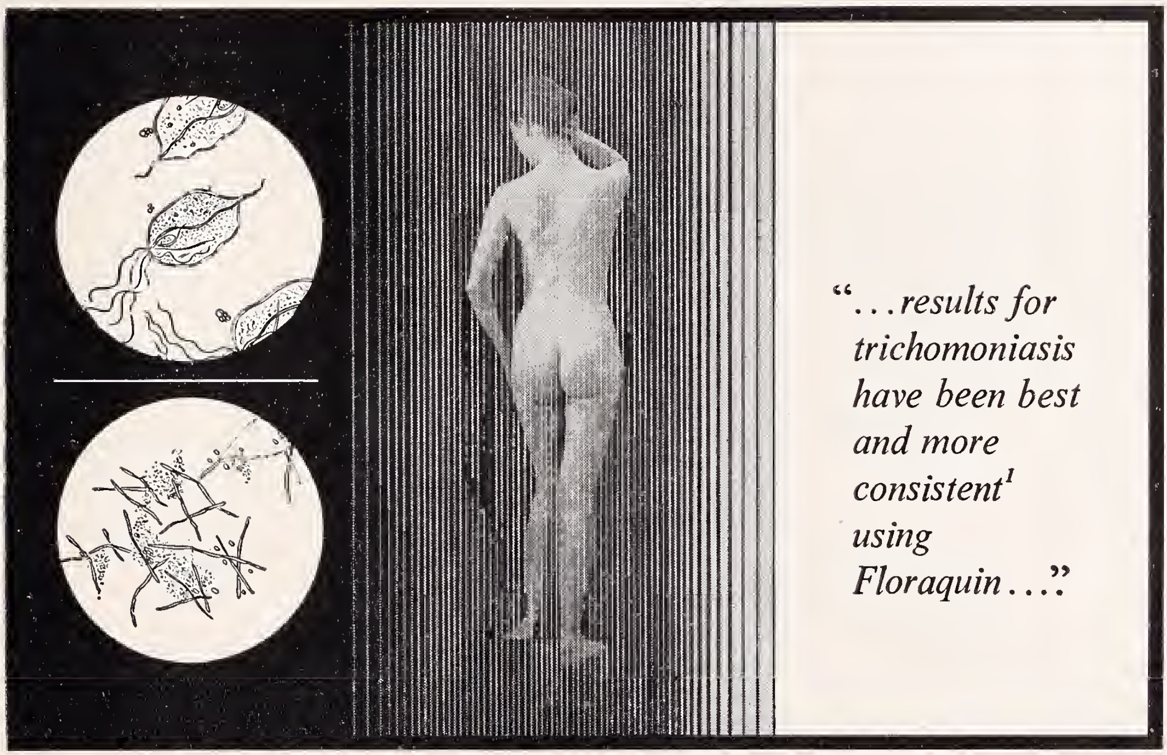
Blue Shield benefits are provided for services reported on well over 90 per cent of the reports submitted. Less than ten per cent of all claims received are rejected or forwarded to other Blue Shield Plans. However, all claims received require at least limited processing.

While the Blue Shield work-load increased greatly during 1957, and again during January and February of 1958, the number of employes has not been increased. Improved methods have enabled the Claims Department to process the increased number of claims, well over 1,000 each working day, without additional help.

During the year 1957, Minnesota Blue Cross provided hospitalization benefits for nearly one out of every two contracts in effect. This experience marked a new high in frequency of subscriber usage of Blue Cross benefits.

(Continued on Page A-42)

EFFECTIVE, DEPENDABLE THERAPY FOR VAGINITIS



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Floraquin® eliminates trichomonal and mycotic infection; restores normal vaginal acidity

Leukorrhea is by far the most frequent symptom of vaginitis; trichomonads and monilia are the most common causes. Many authors have reported[2] trichomonal protozoa in the vagina of 25 per cent of obstetric and gynecologic patients. Increased use of broad spectrum antibiotics has resulted in a sharp rise in the incidence of monilial infections.

Floraquin effectively eradicates both trichomonal and monilial vaginal infections through the action of its Diodoquin® content. Floraquin also furnishes boric acid and sugar to restore the normal vaginal acidity which inhibits pathogens and favors the growth of protective Döderlein bacilli.

Pitt[1] recommends vaginal insufflation of Floraquin powder daily for three to five days, followed by acid douches and the daily insertion of Floraquin vaginal tablets throughout one or two menstrual cycles. G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

---

1. Pitt, M. B.: Leukorrhea. Causes and Management, J. M. A. Alabama *25:*182 (Feb.) 1956.
2. Parker, R. T.; Jones, C. P., and Thomas, W. L.: Pruritus Vulvae, North Carolina M. J. *16:*570 (Dec.) 1955.

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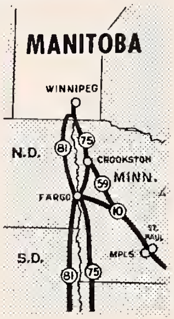

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MINNESOTA BLUE CROSS-BLUE SHIELD

(Continued from Page 290)

January, 1958, experience indicates a continuation of the 1957 high-incidence trend. Over 18,000 subscribers were provided 112,239 days of hospital care during the month. The incidence rate represented is 513 cases paid per year per 1,000 contracts protected—over one out of every two contracts protected—a five per cent increase over the frequency of Blue Cross usage during the previous January. Each hospitalized subscriber stayed an average of 6.2 days, an increase of two-tenths of a day compared with the average length of stay during January, 1957.

Accidental injuries and poisonings led all other reasons for hospitalization of Blue Cross subscribers with 3,784 admissions representing 20.8 per cent of all cases paid during the month of January, 1958. The frequency of incurrence of such cases reached a new high—with 107 cases paid per year per 1,000 contracts protected—a 40 per cent increase in frequency compared with the previous January.

The normal trend of incidence of accidental injuries and poisonings is an increased rate over the summer months of the year as compared with the winter months—rather the reverse of the trend in frequency of respiratory illness occurrence. However, as seen in the chart above, January, 1958, experience exceeds that of the highest peak of the preceding two years' Blue Cross experience.

Pregnancy ranked third in reasons for hospitalization of Blue Cross subscribers with 2,638 admissions representing 14.5 per cent of the cases paid during January, 1958. These cases required 12,453.3 days of hospital care, an average of 4.7 days per case.

Diseases of the respiratory system continue to be a leading factor in Blue Cross utilization. During January, 1958, the second leading cause of subscriber hospitalization was respiratory illnesses. The 2,974 admissions involved representing 16.3 per cent of all cases paid required 14,224.7 days of care, an average of 4.8 days per case.

Although the high frequency of hospitalization for respiratory illnesses is continuing, the incidence rate of 84 cases paid per year per 1,000 contracts protected is not excessively high for this season of the year. It is interesting to note that the average hospital stay has decreased from 5.3 days and 5.5 days per case during November and December, 1957 respectively to 4.8 days per case during January, 1958.

Social security has been changed many times in the twenty-two years since the original law was enacted. The size and variety of benefits, the tax rates, the tax base, coverage—all have been radically changed. There is no reason to believe that another twenty-two years will not see just as radical changes.

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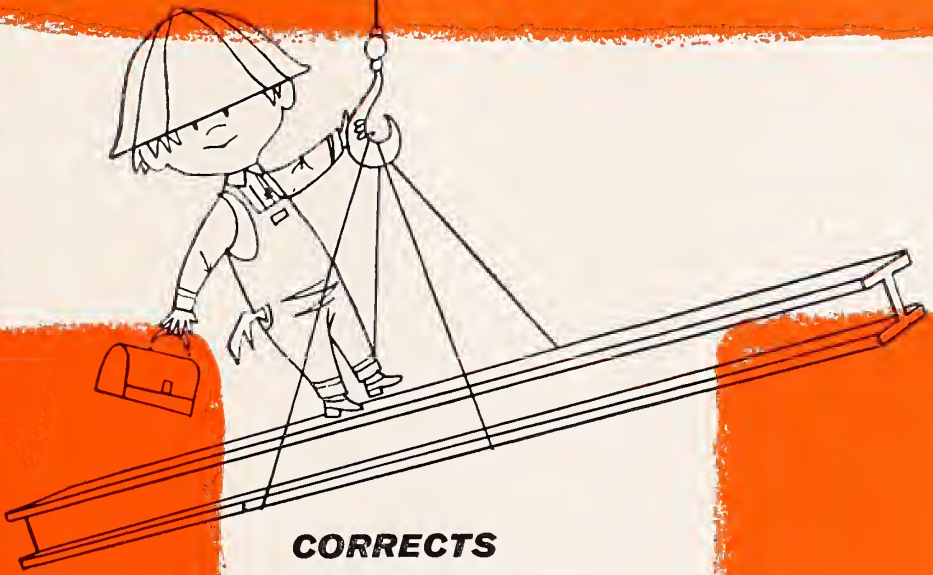

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INCREMIN Syrup

FORMULA: Each teaspoonful (5 cc.) contains:

l-Lysine HCl	300 mg.
Ferric Pyrophosphate (Soluble)	250 mg.
Iron (as Ferric Pyrophosphate)	30 mg.
Vitamin B_{12} Crystalline	25 mcgm.
Thiamine Mononitrate (B_1)	10 mg.
Pyridoxine HCl (B_6)	5 mg.
Alcohol	0.75%

Average dosage is 1 teaspoonful daily.
Available in bottles of 4 fl. oz.

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Meetings and Announcements

STATE

Minnesota Association of Blood Banks, 1958 anuual meeting, in conjunction with the Upper Midwest Hospital Conference, Minneapolis Auditorium, May 14, 1958.

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

Minnesota Surgical Society, spring meeting, Rochester, Minnesota, May 9, 1958. O. H. Beahrs, M.D., Secretary, Rochester, Minnesota.

NATIONAL

American Congress of Physical Medicine and Rehabilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

American Goiter Association, 1958 meeting, June 17-19, San Francisco, California.

First All-Day Forum on Medical Economics, March 27, 1958, Washington, D. C. Co-sponsored by the Medical Council of the Washington Metropolitan Area and the Wm. S. Merrell Company of Cincinnati, Ohio.

Gerontological Society, Inc., Bellevue Stratford Hotel, eleventh annual scientific meeting, Philadelphia, Pennsylvania, November 6, 7, and 8, 1958.

Symposium on "Basal Gangila Surgery for Involuntary Movement Disorders." New York University-Bellevue Medical Center, Thursday, May 15 to Saturday, May 17. Complimentary tickets. Miss Dorothy P. Frome, Office of Public Relations, 550 First Avenue, New York 16, New York.

Trudeau School of Tuberculosis and Other Pulmonary Diseases, forty-third session, Saranac Lake, N. Y., June 2 to 20, 1958. Detailed information may be obtained from the Secretary, Trudeau School of Tuberculosis and Other Pulmonary Diseases, Box 500 Saranac Lake, N. Y.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy, Association, Paris, France, October 19-26, 1958.

World Congress of Gastroenterology, Washington, D. C., May 24-31, 1958. Joint meeting with the American Gastroenterological Association, Dr. H. M. Pollard, Secretary-General, University Hospital, Ann Arbor, Michigan.

LOVELACE FOUNDATION PROGRAM

The Lovelace Foundation, Albuquerque, New Mexico, has instituted a program for Visiting Physicians and Surgeons *pro tem.* The program is intended to further the opportunities for graduate medical education in bringing to practicing physicians, the latest advances of fundamental and clinical medicine and surgery.

The first Visiting Physician *pro tem* was Dr. Arthur M. Olsen, Professor of Medicine, Graduate School, University of Minnesota, and one of the Chiefs of Medicine of the Mayo Clinic.

MEDICAL EDUCATION WEEK

The American Medical Association has announced that it will again join the nation's 83 medical colleges in helping develop and promote the Third Annual Medical Education Week, April 20-26, 1958.

The National Committee on Medical Education Week will send promotional kits to state and county medical societies for use during this week. These kits will contain scripts, speeches, sample proclamations, editorials, and other programs for civic attention.

CONTINUATION COURSES

Medical continuation courses to be presented at the Center for Continuation Study, University of Minnesota are as follows:

April 7-9—Radiology for General Physicians
April 10-12—Arthritis and Physical Medicine for General Physicians
April 12—Trauma for General Physicians
April 14-16—Gastroenterology for General Physicians
May 5-9—Electrocardiography for General Physicians & Specialists
May 12-16—Proctology for General Physicians

For further information concerning the above courses. write to the Director, Department of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14, Minnesota.

ADVANCED ORAL PATHOLOGY COURSE

The Dental and Medical Faculty of the University of Minnesota will present a course in Advanced Oral Pathology during the week of April 28 through May 2, 1958. Both lectures and pathologic slide study will be included. Microscopes will be provided. The material presented will include: neoplastic and non-

(Continued on Page A-46)

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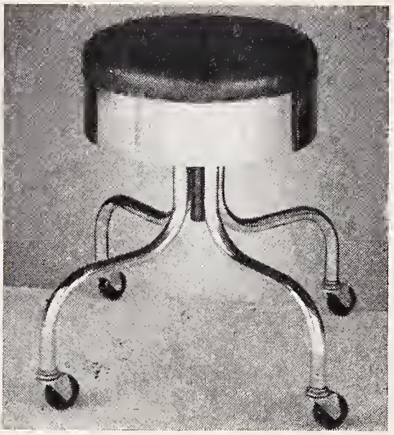

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ADVANCED ORAL PATHOLOGY COURSE

(Continued from Page A-44)

neoplastic diseases of the salivary glands, neoplasms of dental origin, bone pathology, soft tissue neoplasms, dermatologic pathology, diseases of the lymphoid system, and oral roentogenographic evidence of systemic disease.

The faculty will include Dr. Robert Lukes, Armed Forces Institute of Pathology, Dr. D. C. Dahlin and Dr. Edward Stafne of Mayo Clinic, Dr. Robert Goltz of the Medical School, University of Minnesota, Dr. Anand Chaudhry and Dr. Robert J. Gorlin of the School of Dentistry, University of Minnesota.

The tuition will be $50 for the week. If room and board at the Center for Continuation Studies are desired, the cost for the week will be about $50.

WARNING TO MINNESOTA PHYSICIANS

An increasing number of Minnesota physicians are falling prey to unlicensed securities brokers both from Canada and New York and other eastern cities by purchasing securities of unknown value.

Arthur Hansen, Commissioner of Securities, has warned that purchase of unregistered securities offered by these out-of-state and out-of-country confidence men who make contact by "hurry up" literature and long distance telephone calls and telegraph messages invariably results in substantial losses to the investor.

From a recent investigation by the Securities Commission of one of these swindling securities dealers, it was noted that eleven of the twenty-six Minnesota victims were physicians who thus far have suffered losses of more than $18,000. As these losses can be avoided, the Commissioner hopes this warning will be kept in mind when physicians are approached by unknown brokers.

Mr. Hansen's office is always available to answer questions concerning securities and a call or a card, when in doubt, to the Securities Division, 202 State Office Building, St. Paul, will save the prospective purchaser money and disappointment.

GOLF TOURNAMENT

The American Medical Golfing Association is holding its annual golf tournament in conjunction with the AMA convention June 23, 1958, at the beautiful Olympic Lakeside Golf and Country Club, San Francisco, California. This will be a whole day of rest and relaxation with golf, luncheon, banquet, and a prize for everyone. No stone has been left unturned to assure participants the very best. Tee off time: 8 a.m. to 2 p.m. All golfing doctors are cordially invited to attend. Handicaps scratch to 30 in flights.

For information, write James J. Leary, M.D., Secretary, 450 Sutter Street, San Francisco, California.

REGISTERED NURSE LICENSURE CHANGES

The Minnesota Board of Nursing announces a change in policy relative to applications for Registered Nurse licensure by interstate endorsement. A nurse licensed elsewhere may not be employed as a registered nurse in Minnesota until she obtains a Minnesota license as such—except while her application for this license is pending. A "courtesy" status is provided while the application is pending for a period *not to exceed six months from the date the application is filed in the Minnesota Board of Nursing office, 700 Minnesota Building, St. Paul 1.* Persons expecting to be employed as registered nurses in Minnesota should request application forms *shortly before* beginning employment in this state.

LABORATORY ASSISTANTS COURSE

The General Extension Division of the University of Minnesota announces that the next course for laboratory assistants will begin July 1, 1958. The twelve-month program trains qualified high school graduates in limited laboratory techniques in hematology, urinalysis, BMR-EKG tests, chemistry, and blood banking. At the end of this time, the students are best qualified to work in a physician's office or to assist a registered medical technologist M.T. (ASCP) in a hospital or clinical laboratory. Further information and application blanks about the program can be obtained from Margaret Ohlen, Instructor and Co-ordinator, Medical Laboratory Assistant Program, C205 Mayo Building, University of Minnesota, Minneapolis 14, Minnesota.

CAMP PHYSICIANS WANTED

The Saint Paul Area Boy Scout Camp is again requesting the services of interested physicians. One-week periods of camp run from June 14, 1958, to the latter part of August. A new cabin with all modern plumbing facilities is supplied to the doctor and his family. Two nurses are on duty at all times at the camp.

For further information, write or call Dr. Donald W. Koza, 1210 Lowry Medical Arts Building, Saint Paul 2, Minnesota. Telephone: CApitol 4-5371.

SYMPOSIUM ON CARDIOVASCULAR DISEASE

Preceding the dedication of the new Lake Region Hospital in Fergus Falls, Minnesota, the medical staff is sponsoring a symposium on "The Frontiers of Cardiovascular Disease." This is to be held on Saturday, June 28, 1958. The faculty will consist of physicians specializing in cardiovascular disease from Rochester, Minnesota, the Twin Cities, and Winnipeg, Canada. All doctors interested are cordially invited to attend. The program will appear in the next issue.

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Woman's Auxiliary

ANNUAL MEETING PROGRAM HIGHLIGHTS

The thirty-sixth annual meeting of the Woman's Auxiliary to the Minnesota State Medical Association will be held May 22-24 in Minneapolis. Convention headquarters for the centennial year observance will be the Curtis Hotel. Auxiliary convention chairmen are Mrs. Conrad Holmberg and Mrs. Mancel Mitchell.

Several highlights of this year's annual meeting will include the executive board meeting and luncheon for county presidents and presidents-elect to be held Thursday, May 22. Following the luncheon, guests will tour the Hennepin County Historical Museum, and will climax that afternoon's activities with a tea at the Minneapolis Art Institute. Thursday evening members of the auxiliary and their husbands have been extended invitations to be the guests of the Hennepin County Medical Society at special entertainment planned for the occasion. The annual meeting and luncheon for all doctors' wives is scheduled for Friday noon, May 23. The annual banquet of the Minnesota State Medical Association will be held Friday, May 23, at 7:00 p.m. in the Leamington Hotel in downtown Minneapolis. Committee chairmen for the event stress the importance of making early reservations for the various events of the annual meeting.

NATIONAL AND INTERNATIONAL MEETINGS

The national meeting of the Woman's Auxiliary to the American Medical Association will be held this year in San Francisco, June 25-27. Immediately following the A.M.A. annual meeting in San Francisco, the Hawaii Summer Medical Conference will be held in Honolulu, July 1-3, 1958.

ST. LOUIS COUNTY AUXILIARY

Recent activities of the St. Louis County Medical Auxiliary include:

Presentation of ten dollars to the winner of the Essay Contest sponsored by the Tuberculosis Society.

Awarding of two nurses scholarships of two hundred dollars each.

Presentation of the invitational annual dinner dance for doctors and their wives from Superior, Wisconsin.

Inauguration of a new volunteer service at one of the hospitals—stork photography. In addition, Auxiliary members are serving as volunteers in related public service areas.

AUXILIARIES SPONSOR MEDICAL VOCATIONS TEAS

Mower County Auxiliary

Members of the Mower County Medical Auxiliary were hostesses at a tea January 24, 1958, for sophomore girls of all Mower County high schools interested in nursing or affiliated fields of the medical profession. Mrs. Wallace Anderson, Auxiliary president, was chairman and introduced the speakers for the nursing field; Mrs. Virginia Spongler, Macalester College, and three Minneapolis student nurses, Lois Barta, Abbott Hospital; Marlene Thimigau, St. Barnabas Hospital; Mrs. Alice Peterson, Northwestern Hospital.

Mower County Auixilary members representing medical technology are Mrs. E. C. Sargent, Mrs. James Thomson, and Mrs. R. R. Wright.

Stearns-Benton County Auxiliary

High school girls from the St. Cloud area were guests recently at a nurses' tea sponsored by the Stearns-Benton Medical Auxiliary in the St. Cloud Hospital School of Nursing home. Those attending included: Mrs. C. F. Brigham, Jr., Arleen Dufault, Patricia Murphy, Mary Kay Ployhart, Carol Schwartz, Mary Kay Popp, Judy Maslonkowski, Jean Smoley and Sister Annora, OSB, assistant administrator.

Must all cardiac patients cut down drastically on exercise? Studies show that over 30 per cent of them require no restriction of activity whatever. Approximately another 40 per cent must avoid only severe exertion. In fact, less than 1 per cent require "complete rest."—*Patterns of Disease.*

* * *

Beer may act favorably in heart failure and arterial hypertension and is a welcome addition to a diet poor in salt, says Dr. B. Josephson of St. Erik's Hospital, Stockholm. Patients there are permitted from 300 to 600 milliliters (approximately one to two pints) of beer daily. He says the method has had similar results in Milwaukee, U.S.A. "No comment!" remarks Dr. Morris Fishbein.—*Medical Science,* December 10, 1957.

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THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

LICENSE OF RED WING PHYSICIAN SUSPENDED

The Minnesota State Board of Medical Examiners, on February 15, 1958, suspended for a period of three years the license to practice medicine held by Dr. Howard F. Claydon, fifty-two years of age, of Red Wing, Minnesota. Dr. Claydon had previously been charged in a citation issued by the Board with immoral, dishonorable and unprofessional conduct, as defined by law, in that he had procured, aided and abetted a criminal abortion upon a Minneapolis woman. Dr. Claydon had appeared at a prior meeting of the Board and admitted the truth of the charge in the citation, but because Dr. Claydon was undergoing treatment by a physician for a very serious ailment, the Minnesota State Board of Medical Examiners took the matter under advisement at that time and deferred making a decision in regard to the disciplinary action that should be taken.

An investigation by an agent of the Minnesota Bureau of Criminal Apprehension and a representative of the Minnesota State Board of Medical Examiners disclosed that a thirty-five-year-old Minneapolis divorcee, after learning from an examination by a Minneapolis physician that she was pregnant, was driven to Red Wing by a woman companion who pointed out Dr. Claydon's office to her. The patient, in a signed statement, indicated that she paid $250.00 in cash to Dr. Claydon for performing an abortion upon her, and Dr. Claydon admitted that this was true when he appeared before the Board in response to the citation. After returning to Minneapolis, the patient became ill and was hospitalized in a Minneapolis hospital where she passed the fetus.

Dr. Claydon, who was born in Red Wing, Minnesota, on October 10, 1905, graduated from the University of Louisville, Louisville, Kentucky, with an M.D. degree in 1929. Dr. Claydon was licensed to practice medicine in Minnesota on December 2, 1930 by reciprocity with Kentucky. Dr. Claydon then entered the practice of medicine at Zumbrota, Minnesota, and remained there until 1947 when he moved to Red Wing.

NEW ULM WOMAN SENTENCED

On March 1, 1958, Marlys Posusta, twenty-seven, New Ulm, Minnesota, was sentenced in that city by the Hon. George D. Erickson, Judge of the District Court, to serve a term of from one to four years in the State Reformatory for Women at Shakopee. The defendant had previously on January 24, 1958, entered a plea of guilty before Judge Erickson to the crime of wilfully producing her own miscarriage and at that time the Court continued the case and ordered that a pre-sentence investigation be made. Judge Erickson granted a stay of execution of the sentence until March 7, 1958, in order to give the defendant time to arrange her personal affairs, but he stated that under the circumstances he did not feel that he would be justified in placing the defendant on probation.

The original investigation in this case, which was conducted by an agent of the Minnesota Bureau of Criminal Apprehension and a representative of the Minnesota State Board of Medical Examiners, disclosed that the defendant had aborted as a result of taking medication and using an instrument upon herself. Miss Posusta became ill after passing the fetus in a friend's apartment in Minneapolis and it became necessary for her to be hospitalized in a Robbinsdale hospital.

When questioned by the Court, the defendant said that she was born on July 26, 1929, at Silver Lake, Minnesota, and that she never married. Miss Posusta, who has no training in any of the healing arts, further stated that she did not attend school beyond the ninth grade but that she had worked in a canning factory at Winsted and had been employed as a waitress in Glencoe.

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In Memoriam

RICHARD E. POGUE

Dr. Richard E. Pogue, former Watertown physician, died February 22, 1958, in Carson City, Nevada. He was fifty-three years old. Doctor Pogue was a practicing physician in Watertown for approximately twenty-three years. He retired in 1957.

A native of Duluth, he received his preliminary education at Maplewood Academy. He attended Union College, Lincoln, Nebraska, and Pacific-Union College, Angwin, California.

His medical education was obtained at the College of Medical Evangelists, Loma Linda, California. His special work included internship at Orange County Hospital, Santa Ana, California.

Positions and professional affiliations held by Dr. Pogue include: Superintendent, Cottage Hospital, Watertown, Minnesota; Staff Member, Fairview Hospital and Swedish Hospital, Minneapolis.

He was a member of the Scott-Carver County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

He is survived by his wife, Viola; a son, Richard, Jr., and a daughter, Patricia, all of Watertown.

DONALD R. READER

Dr. Donald R. Reader, St. Paul physician, was killed in an automobile accident February 20, 1958 when his car missed a curve on highway 52, near Dalton, Minnesota. He was forty-six years old.

A native of St. Paul, he received his preliminary education at Cretin High School.

Dr. Reader was a 1958 graduate of the University of Minnesota Medical School. His special work included twelve months' internship at St. Mary's Hospital in Minneapolis.

Dr. Reader was a member of the Incus, honorary medical fraternity; Minnesota Neurological Society, American Psychiatric Association and a diplomate of the American Board of Psychiatry. He was associated with Glenwood Hills Hospital and the mental hygiene section of Veterans' Hospital.

In addition, he was an active member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife Mary; two children, Joanna, seventeen, Thomas, thirteen, and two brothers, Lawrence, Malvern, Pa., and Mark, Minneapolis.

TAKE A LOOK AT
NEW DIMETANE
THE UNEXCELLED
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MAGNUS WESTBY

Dr. Magnus Westby, a practicing physician in Madison, Minnesota, died March 13, 1958, while vacationing in Santa Ana, California, at the age of fifty-eight.

In 1955, Dr. Westby was appointed to the State Board of Medical Examiners by Governor Orville Freeman. He held the post as chairman of that board at the time of his death.

A native of Aberdeen, South Dakota, he graduated from St. Olaf College, Northfield, Minnesota, and from University of Minnesota Medical School in 1924. In 1925, he began practicing medicine in Madison.

Dr. Westby served two terms as mayor of Madison. He was a member of the American Legion, Madison Masonic Lodge, Phi Beta Pi medical fraternity, Minneapolis Athletic Club and Faith Lutheran church, Madison. He also was past president of Camp Release Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include two sons, John and Robert, Minneapolis, and five brothers, Sigurd, Willmar; Edmund, Sioux Falls, S. D.; Fritjof, Aberdeen, S. D.; Carl, Culbertson, Mont., and Dr. Peter Westby, Madison.

Forty years ago, according to Health Information Foundation, one in every ten babies born alive in this country was unable to survive the first year of life. Today, the ratio has dropped to one in forty.

Correspondence

To the Editor:

Attention is called to the president's letter in this issue which deals with the Physician's Assistance Committee. I have been deep in organization work during my entire medical career and I believe that this committee is of the utmost importance. I prize most my service as a delegate and I believe the delegate to the House of Delegates of the State Medical Association is the most important man in medicine today. Next I prize most my appointment to the Physician's Assistance Committee. Some committees work hard, some do nothing; some committees accomplish much, some nothing. The Physician's Assistance Committee is working hard and is doing more good for the average practitioner in Minnesota than ever will be appreciated.

Yours truly,
James A. Blake, M.D.

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* * *

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General Interest

A Duluth physician has been honored by the Minnesota Heart Association. **Dr. F. J. Hirschboeck**, one of the founders and former president of the organization, is the recipient of the distinguished service award for outstanding contributions in the field of cardiac health. The award, first ever made by the association in its ten-year history, was presented at the Duluth Covenant Club at a kick-off dinner for the annual Duluth Heart Fund campaign.

* * *

Dr. Alfred Doscherholmen, of the Minneapolis VA hospital, is one of eight recent clinical investigators appointments announced by the Veterans Administration.

* * *

Dr. C. J. Corrigan has joined the consulting staff of the Zumbrota Community Hospital as x-ray specialist. He is also a member of the staff of Olmsted County Hospital in Rochester.

* * *

Dr. F. Henry Ellis, Mayo Clinic surgeon, recently participated in a panel discussion on esophagitis at the sectional meeting of the American College of Surgeons held in New York City.

* * *

Dr. Harold O. Peterson was elected a member of the Board of the American College of Radiology at the annual meeting in Chicago. Dr. Peterson, a graduate of the University of Minnesota and the University's Medical School, will serve four years as a member of the Board of Chancellors.

* * *

Dr. Louis A. Buie, emeritus head of the Section of Proctology of the Mayo Clinic and emeritus professor of proctology in the Mayo Foundation, has been named secretary-treasurer of the Advisory Board for Medical Specialties. Dr. Buie had been acting secretary and treasurer of the board since the death of **Dr. B. R. Kirklin** on March 2, 1957. He has been a member of the board since 1949.

* * *

"The Heart—Its Functions and Malfunctions," was the title of a recent address presented by **Dr. Richard A. DeWall** at a recent St. Paul Junior Chamber of Commerce luncheon.

* * *

At the annual meeting of the medical staff of St. Barnabas Hospital, **Dr. Edgar A. Webb** was elected chief-of-staff, succeeding **Dr. William T. Walsh.** The new Vice chief-of-staff is **Dr. C. R. Peluso** and the secretary is **Dr. D. G. Bohn.**

* * *

Several Rochester men were participants in the 11th biennial International Congress of the International College of Surgeons, March 9-13, in Los Angeles in conjunction with the 23rd annual Congress of the United States and Canadian Sections. **Dr. Henry W. Meyerding**, an emeritus member of the Mayo Clinic staff, is president-elect of the International College. He delivered a paper on "Errors in Treatment of Fractures of Upper Extremities and Their Consequences." **Dr. Winchell Craig**, emeritus Mayo Clinic staff member, and **Dr. Gershom Thompson**, of the Clinic staff, were on the special panel on "Management of Surgical Emergencies." Moderator was **Dr. Ross T. McIntire**, former Surgeon-General of the Navy, now executive director of the International College of Surgeons. Dr. Craig discussed "Injuries of the Head and Spine" and Dr. Thompson talked on "Injuries of the Bladder and Genitalia." Also on the program from the Clinic were **Dr. John Henderson**, moderator of a panel on Symposium on Procedures in Surgical Ophthalmology, and **Dr. A. M. Olsen**, who took part in a symposium on "The Inter-Relation of Pulmonary and Esophageal Diseases."

* * *

Two staff members of the Mayo Clinic were among speakers at the annual clinical conference of the Chicago Medical Society in Chicago. They were **Dr. Hamilton Montgomery**, senior consultant in the section of dermatology, and **Dr. Arthur B. Hunt** of the section of obstetrics and gynecology.

* * *

The new Wright County Medical Advisor is **Dr. G. Harland Purves**, of Buffalo. He succeeds **Dr. Robert M. Sandeen** of that community.

* * *

"Of Thee I Sing; Of Me I Give," was the topic of a recent University of Minnesota Greek Week banquet address given by **Dr. Edward H. Rynearson**, chairman of the section of metabolic diseases at the Mayo Clinic.

* * *

Dr. J. P. Grahek, Ely mayor, has been appointed to the position of the first chief of staff of the medical staff of the Ely-Bloomenson Community Hospital. The announcement was made at the first annual meeting of the shareholders of that corporation. Recently appointed to the staff were **Dr. Edward Ciriacy** and **Dr. Robert Kundel.**

* * *

Three Mayo Clinic staff members recently participated in an international symposium on "Pulmonary Circulation," sponsored by the Chicago Heart Association in the Palmer House at Chicago, March 20-22. They were **Drs. Howard B. Burchell, Jesse Edward** and **John Shepherd.**

* * *

Dr. Douglas L. Johnson has returned to Little Falls following a three-year residency in ophthalmology at the University of Minnesota hospitals. He announced the opening of offices at the Medical Center in Little Falls and at the O'Brien Center in Brainerd. Dr. Johnson's practice will be limited to diseases of the eye and eye surgery. He was formerly in general practice in Little Falls from 1937 to 1955.

(Continued on Page A-56)

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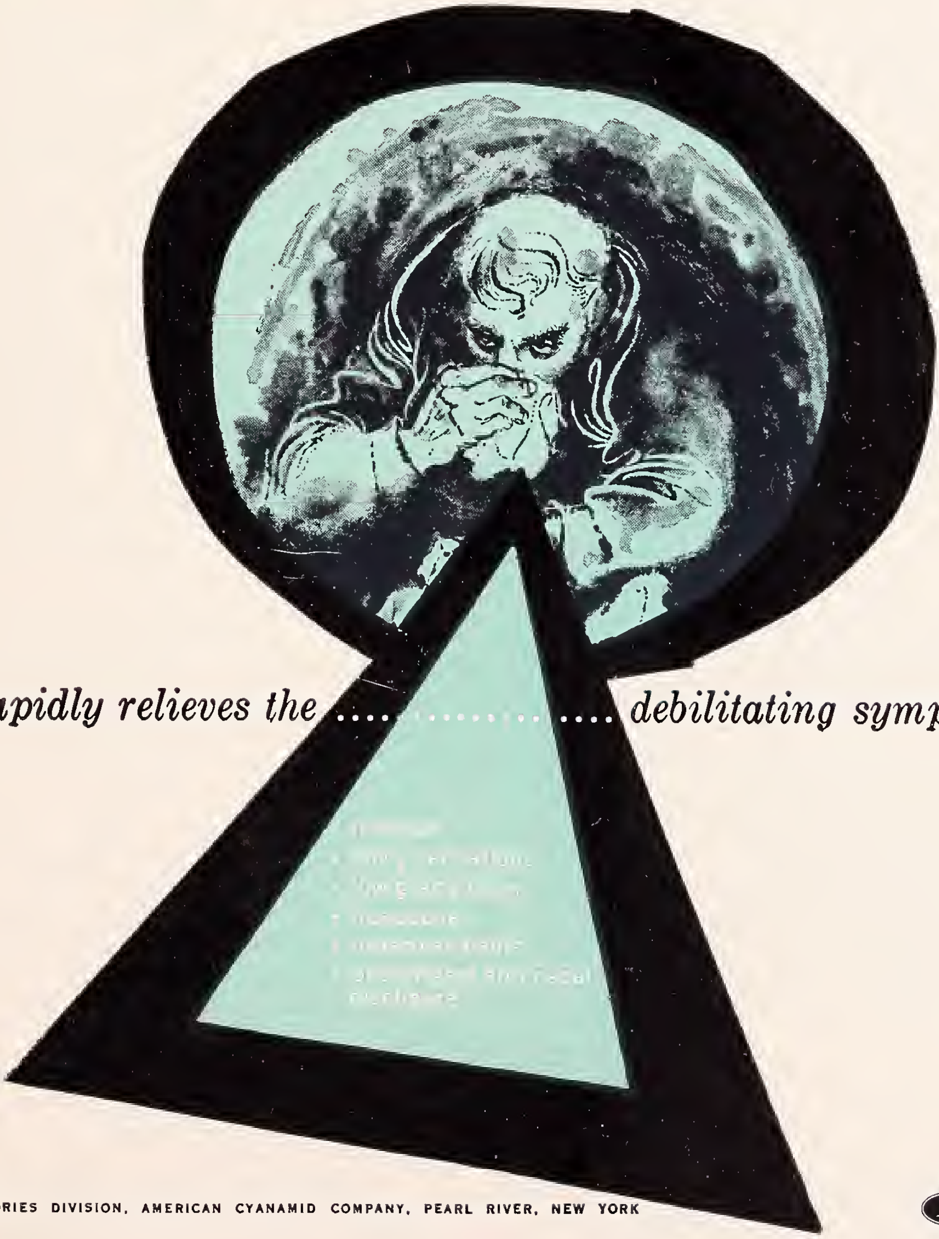
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(Continued from Page A-54)

Members of a recently appointed Poison Center Advisory Committee included the following representatives of state medical and lay groups. Committee members are: **Dr. Frank A. Ubel,** Ramsey County Medical Society; Boris L. Levich, St. Paul Department of Public Safety; **Dr. W. E. Parks,** Minneapolis Division of Public Health; **Dr. Donald E. Roach,** Minnesota Academy of General Practitioners; Glenn I. Prickett, Minnesota Safety Council; Henry Moen, Minnesota State Pharmaceutical Association; **Dr. James Fox,** Minnesota Academy of Occupational Medicine and Surgery; George S. Michaelson, Minnesota Hospital Association, Inc.; **Dr. Tague Chisholm,** Minnesota Academy of Medicine; Mrs. Richard Angevina, Minnesota Congress of Parents and Teachers, Inc.; **Dr. Raymond N. Bieter,** head of the department of pharmacology, University of Minnesota School of Medicine, **Dr. Harold N. Wright,** professor of pharmacology on the University staff, and Harold Brunn, Minnesota State Medical Association.

* * *

Dr. Edward A. Meyerding, well known in the state and nation for outstanding contributions to the cause of public health, retired in April as Managing Director of the Minnesota Tuberculosis and Health Association.

During the first thirteen years of his more than a third of a century as executive of the state Christmas Seal organization, Dr. Meyerding also served as secretary of the Minnesota State Medical Association. Dr. Meyerding resigned from the Medical Association position to devote his entire time to the tuberculosis program.

With the aid of his staff, Dr. Meyerding has built up an organization with approximately 10,000 volunteer workers and many thousands of interested contributors. People in all counties of the state are informed about tuberculosis and health problems through newspaper stories, radio and TV information, literature, conferences, and special courses and through *Everybody's Health,* Christmas Seal magazine.

Under his leadership, the state Christmas Seal organization since 1930 has helped to finance hundreds of tuberculin testing surveys in schools. Mantoux testing, emphasized as the major case-finding program, has in recent years been extended to adults as well as children in community-wide and county-wide surveys.

The Christmas Seal organization has worked closely with the State Medical Association and the Minnesota Department of Health in the accreditation of counties for progress in reducing tuberculosis death and infection rates; in tuberculin testing and in mass x-ray surveys; and in promoting the routine x-raying of hospital admissions.

Dr. Meyerding's leadership and his ability to adapt the program against tuberculosis to changing methods and media have been important factors in placing Minnesota in the outstanding position it holds nationally in the sale of Christmas Seals and in carrying out the program they finance.

During the thirty-four years that he has headed the state Christmas Seal organization, he has seen the tuberculosis death rate in Minnesota drop from 69.5 per 100,000 population in 1924 to 3.6 in 1956.

* * *

Dr. Wade R. Humphrey, of Stillwater, has resigned as chairman of the Physician's Assistance Committee of the Minnesota State Medical Association because of ill health and has been replaced by **Dr. James A. Blake,** of Hopkins, who has been pinch-hitting for Dr. Humphrey as vice chairman for the past year. (See the President's message in this issue).

* * *

Dr. Gershom J. Thompson has been elected a vice president of the United States section of the International College of Surgeons.

* * *

Dr. Milton L. Kaiser, thirty-four, has been named New Ulm's outstanding young man of 1957. The selection was announced at the annual Distinguished Service Award banquet and Bosses Night program at Turner Hall. About 100 persons attended the annual Junior Chamber of Commerce project.

* * *

Dr. H. P. Linner, Minneapolis physician and surgeon, is in Temple Hospital, Philadelphia, where he underwent brain surgery to alleviate a nervous illness. Dr. Linner gave up active practice a year and a half ago because of Parkinson's disease. In recent years, his sons, Dr. John and Dr. Paul, have taken over most of his work.

* * *

Four new members of Swedish Hospital's board of trustees have been elected. They are **Dr. John S. Milton, Dr. Leonard Kendall,** C. Albert Carlson and Edward H. Norblom.

* * *

Dr. David H. Kaplan has been named chief medical

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(*Name of university and results of clinical tests sent upon written request.)

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Milk protein	2.60%
Milk sugar (lactose)	4.20%
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Milk minerals	.62%
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Iron	5.6 mg. per qt.
Water	88.0%

VITAMIN CONTENT

	Per Qt.
Vitamin A (U.S.P. units)	3000
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Vitamin C (ascorbic acid)	100 mg.
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officer of the Veterans Administration center at Fort Snelling. Kaplan, fifty-two, succeeds **Dr. Richard B. Hullsiek** who died February 1. Kaplan has been assistant chief medical officer at the center since 1946.

* * *

Dr. C. Walton Lillehei, University of Minnesota heart surgeon, has been chosen 1958 recipient of the Oscar B. Hunter Memorial Award of the American Therapeutic society.

The award will be conferred upon Dr. Lillehei at the society's forty-ninth annual meeting in San Francisco in June. Presentation will take place June 21, when Dr. Lillehei will address the society on his world-famous accomplishments in the repair of congenital and acquired heart defects by open heart surgery.

* * *

Dr. C. A. Saffert, president, presided at the quarterly seminar of the southern Minnesota regional chapter of the American Academy of General Practitioners at the Colony Club in New Ulm.

Speakers included **Dr. Walter P. Eter,** whose topic was "General Management of the Severely Injured Patient"; **Dr. Malcolm McCannel,** speaking on "Management of Eye Injuries"; **Dr. W. Robert Schmidt,** who chose "Management of Trauma to the Chest" as his subject; and **Dr. Lester W. Carlander,** who discussed "Management of Hand Injuries." The seminar for the southern Minnesota group was sponsored by the American College of Surgeons.

Drs. Albert and Carl J. Fritsche of the Fritsche Clinic, New Ulm, has announced the association of two more medical doctors with the clinic. They are **Drs. Peter J. Kitzberger** and **William J. Muesing,** both of New Ulm. They will have offices in the Fritsche Clinic Building, 101½ N. Minnesota. Dr. Kitzberger is a diplomate of the National Board of Medical Examiners and a member of the Minnesota Obstetrical and Gynecological Society.

Following his naval service, Dr. Muesing took four years' training as a surgical resident in the Youngstown, Ohio, Hospitals Association. He returned to New Ulm to practice with the Seifert Clinic.

* * *

Dr. John Lillie, Mayo Clinic, Rochester, Minnesota, will present a paper entitled "Thyrotomy—Technique" at the American Medical Association convention in San Francisco, June 23-27.

* * *

Dr. Gordon R. Kamman, St. Paul, addressed the Nicollet-LeSueur Counties Medical Society in St. Peter, Monday evening, March 10. His subject was "Anxiety and Depression."

* * *

Mayo Clinic staff members attending medical meetings throughout the U. S. during the past few weeks.

Dr. J. B. Brich attended a meeting of the American Fracture Assn. at St. Louis, Mo. He is a member of the board of governors of the Association.

A paper was presented by **Dr. E. V. Allen** at the postgraduate course of the American College of Physicians at Cornell University Medical School at New York.

Dr. R. K. Winkelmann presented a paper at the Second Conference on Progress and Potentials in Leprosy Investigation, held at Carville, La.

Those attending the meeting of the American Orthopsychiatric Assn. in New York City were **Dr. E. M. Litin** and **Dr. G. M. Duncan.**

* * *

"Public Health Horizons in Ramsey County" was the theme for this year's joint meeting of the area's health, welfare, planning and co-ordinating councils.

The annual meeting, held jointly by the four councils of the Greater St. Paul Community Chest and Council, Inc., was attended by over 300 professional and lay men serving the four groups.

Highlights of the meeting was a panel discussion on county health problems. The discussion was moderated by **Dr. F. M. Owens, Jr.,** and was conducted by prominent city and county health officials and members of the League of Women Voters.

* * *

Dr. and Mrs. Frederick A. Figi, Rochester have returned from a round-the-world trip of four and one-half months. During the trip Dr. Figi, emeritus member of the staff of the Mayo Clinic and former head and senior consultant of the section of plastic surgery and laryngology, gave several talks at surgical meetings and universities.

* * *

A community-wide recognition dinner honoring **Dr. Richard A. DeWall** of Minneapolis, formerly of Morris, was held recently at the West Central School of Agriculture

Dr. DeWall's friends and former neighbors in Morris joined in the affair to pay triubute to the thirty-one-year-old research specialist whose development of a simplified mechanical heart-lung machine for use in heart surgery resulted in his selection as one of the Ten Outstanding Young Men of America for 1957 by the United States Junior Chamber of Commerce.

The dinner was sponsored by the Morris Lions Club. Invited to join the Lions were members of Kiawanis Club and the Morris Junior Chamber of Commerce, who originally nominated Dr. DeWall for the TOYM award. Wives of members of the various organizations were guests on this occasion.

* * *

Dr. David B. Robinson, a member of the Section of Psychiatry of the Mayo Clinic since October 1, 1957, will leave Rochester about June 15 to join the department of psychiatry of the State University of New York Medical School, Syracuse, N. Y.

* * *

Faculty of the Mediclinics of Minnesota, held March 2-12, 1958, in Fort Lauderdale, Florida, included: **Dr. Harold F. Buchstein,** Mr. Thomas P. Cook, **Dr. Harry B. Hall, Dr. Arthur C. Kerkhof, Dr. Francis W. Lynch, Dr. Ames W. Naslund, Dr. O. L. Norman Nelson, Dr. Owen F. Robbins, Dr. Albert V. Stoesser, Dr. Robert J. Tenner,** and **Dr. Richard L. Varco.** This year's postgraduate Medical Refresher Course was sponsored by the Florida Academy of General Practice and North Broward General Hospital.

* * *

Following are the 1958 officers of the Rice County Medical Society: President, **Dr. J. G. Beaton,** Northfield; Secretary-Treasurer, **Dr. Thorsten Smith,** Faribault; Vice President, **Dr. Wm. Furlow,** Faribault; Delegate, **Dr. Donald J. Studer,** Faribault; Alternate Delegate, **Dr. J. J. Kolara,** Faribault; and Censors, **Dr. P. G. Bauer,** Faribault; **Dr. Robert Meyer,** Kenyon, and **Dr. D. H. Petersen,** Northfield.

* * *

Dr. J. B. Beaton, Northfield internist, has been acting medical director of the Mineral Springs Tuberculosis Sanatorium at Cannon Falls since the resignation of **Dr. Ezra Bridge.** The former medical director has entered private practice in Port Huron, Michigan.

* * *

Retirement of three Mayo Clinic staff men, **Drs. Ralph K. Ghormley, Hiram E. Essex,** and **James F. Weir,** April 1, has been announced. Dr. Ghormley, an outstanding orthopedic surgeon and former head of the section of orthopedic surgery, has been at the Clinic since September, 1929. He and his wife will settle at Carmel, California, but he does not plan to continue a practice. Dr. Weir, who has been a member of the Clinic staff thirty-three years, is a former president of the staff and former chairman of four sections of medicine, with special interest in diseases of the stomach and intestines, widely recognized for his skill in physical diagnosis and particularly for his clinical work in diseases of the liver. He and Mrs. Weir will continue living in Rochester. Dr. Essex, co-chairman of the Section of Physiology and internationally known for his research work in physiology and experimental medicine, has been at the Clinic since July, 1928. He and his wife will continue living in Rochester.

* * *

Dr. Frank H. Krusen, head of the physical medicine and rehabilitation section of the Mayo Clinic, was a recent speaker at the tenth anniversary celebration of the graduate program in hospital administration sponsored by St. Louis University and the Catholic Hospital Association of the United States and Canada. He discussed "The Patient as an Individual." The entire program of the celebration was devoted to discussion of the hospital needs in an era of change in both its medical and socio-economic aspects.

* * *

Dr. and **Mrs. David B. Robinson** were featured soloists in a recent presentation of "A German Requiem" by Brahms by the Rochester Symphony Orchestra and Oratorio Society in the Mayo Civic Theater.

* * *

Dr. J. Arnold Bargen, chairman of the section of gastroenterology in the Mayo Clinic, and professor of medicine in the Mayo Foundation, has been appointed to the medical advisory board of the National Foundation for Research in Ulcerative Colitis, of Washington,

D. C. The new medical research foundation was established recently in Washington at the end of a nationwide conference on "New Frontiers in Ulcerative Colitis," sponsored by the General Medicine Study Section of the National Institute of Health and supported by a research grant from the new foundation and from the National Institute of Arthritis and Metabolic Diseases, U. S. Public Health Service.

* * *

Drs. Truman and **Homar H. Hedemark,** Ortonville, announced the association of **Dr. George V. Martin** with them in the practice of medicine and surgery at the Hedemark Clinic.

* * *

Dr. O. Theron Clagett, head of a section of surgery in the Mayo Clinic and professor of surgery in the Mayo Foundation, was elected to honorary membership in the Irish College of Surgeons at the Charter Day meeting held in Dublin, February 15. At the meeting he also delivered the Abraham Colles Lecture, named for the celebrated Irish surgeon, who was president of the College of Surgeons before he died in 1843.

* * *

Dr. Jan H. Tillisch of the Mayo Clinic staff was one of nineteen American educators and businessmen who served as the Air University's Board of Visitors in conducting the fourteenth examination of the Air Force's professional education system at Maxwell Air Force Base, Montgomery, Alabama, recently.

DOCTOR'S HOME BECOMES MELTING POT

A Saint Paul physician's home is turning out to be a strange melting pot of Japanese and American cultures. American culture is rubbing off on Hisachika Hirose, seventeen, a Japanese exchange student at Sibley high school, while customs of his native land are making an equally strong imprint on the family of **Dr. and Mrs. M. B. Sorem,** 606 Sibley Hwy., Mendota Heights. Hisachika, a bespectacled lad with a ready smile and ingratiating manners, is making his home with the Sorems while studying as a senior at Sibley high. He's taking to the American tempo and ways of doing things like the proverbial duck to water. A bright student Hisachika is earning straight A's at school despite the linguistic barrier and he's making an equally smooth adjustment to other phases of American living.

Hisachika, whose only apparent difficulty here lies in finding the rare thing that he doesn't like, is particularly captivated with the informality of American schools. He's even critical of the way things are done in Japan when he compares the two educational systems. "Teachers are very friendly in the classrooms here," declared Hisachika, "and they discuss a wide range of subjects outside the course itself. However, in Japan the schools are too formal, with everything too rigid. Relations between teachers and student are never friendly." Another difference appealing to the Japanese youth, whose home is a two hour train ride east of Osaka, is the relatively easier American school work

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load. Hisachika disclosed there is much more emphasis on science and languages in Japan than in America.

For example, Hisachika started studying English in the seventh grade in Japan and had five years of the subject before coming to the U.S. as an exchange student last August. Students wear standard uniforms in Japan, as compared with the great variety of dress used here and little attention is paid to extra-curricular activities. School spirit is practically non-existent in the Japanese high school as compared with its American counterpart, according to Hisachika. "I was surprised when I first came here to see all girl cheerleaders," said Hisachika. "In Japan, girls are too quiet to be cheerleaders. Anyway, there are no cheerleaders in Japan." Elvis Presley and rock and roll are popular with Japanese teen-agers but ballroom-style dancing is practically unheard of except in the larger cities, Hisachika revealed. Hisachika is fitting like a glove into the pattern of American living—both at school and in the Sorem home. The Sorems regard him as one of the family and Hisachika does, too. He constantly refers to Mrs. Sorem as his American "mother."

Although he is scheduled to become a Shinto priest when his grandfather dies, Hisachika has a keen interest in Christianity and attends Hamline Methodist church regularly on Sundays with the Sorems. Hisachika shares a bedroom, chores and other facets of family life with the Sorems's son, Anthony, seventeen, a junior at Sibley high and the two youngsters "are devoted to each other," according to Mrs. Sorem. But it's not entirely a question of Hisachika adjusting to American ways, and the Japanese lad has introduced more than a few innovations in his new American home.

"He's got us eating with chopsticks," smiled Mrs. Sorem, by way of emphasizing that point . . . "Hisachika cooked sukiyaki for us recently and we all thought it was wonderful." Sukiyaki is comparable to American beef stew—with strips of beef and vegetables cooked together. Hisachika is one of a group of forty-nine Japanese students who flew to the U.S. last August to attend American high schools in a program sponsored by American Field Service. Five of the forty-nine Japanese students are attending school in Minnesota. Mrs. Sorem, a member of the American Field Service committee in the St. Paul area, said the foreign students do not come here to be honorary guests in an American home. "These students come here," she continued, "to learn about America and the community in which they live. When they return to Japan next July they will be ambassadors of good will."

She said there is a great need for families in this area to take one of these foreign exchange students into their homes "as an investment in the future." The exchange program provides for American high school students going to foreign countries during the summer months to live with families of those countries. And Mrs. Sorem is convinced that the program is an important instrument in the promotion of world peace. (This news feature appeared in the *St. Paul Dispatch*, Friday, Feb. 1, 1958).

Book Reviews

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MODERN PERINATAL CARE. By Leslie V. Dill, M.D., F.A.C.S., Diplomate of the American Board of Obstetrics and Gynecology. 309 pages. Illus. Price $6.50. New York: Appleton-Century-Crofts, Inc., 1957.

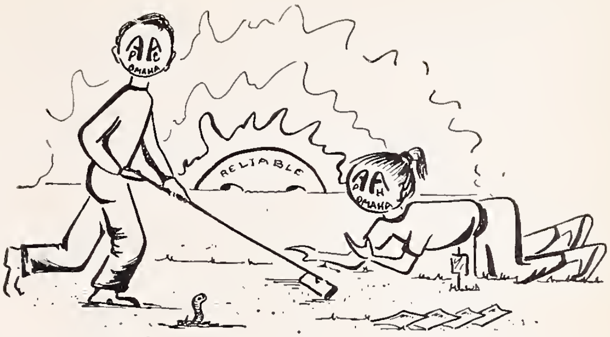
According to the preface, this book has as its aim the crystalization of present-day thought on antepartum and postpartum care. The word "modern" in the title in this case denotes "current." In addition, this book was designed for the utilitarian purpose of giving aid to those practitioners for whom consultation is not readily available.

The book is divided into twenty-one chapters which, among others, include: Manifestations of Pregnancy, Hygiene and Nutrition of Pregnancy, Abortion, Pelvic Mensuration, Psychology of Pregnancy as well as chapters on the common medical complications of pregnancy, the fourth stage of labor, fetal and neonatal mortality, erythroblastosis, infant feeding and obstetric records. In addition, there is a chapter on the practice of obstetrics and the law written by Professor Thomas O'Connell of the Law School of Georgetown University which seeks to define the areas in which malpractice and other legal complications are most likely to arise in obstetric practices. There is also a chapter on the ethics of the Catholic Church pertaining to obstetrics, which was reviewed and edited by three authorities from the Catholic Church. This chapter is especially welcome since there seems to be considerable confusion in the minds of many practitioners, both inside and outside the Catholic Church, regarding the ethics of the Church toward problems involving the care of the pregnant woman.

As is to be expected from a book which seeks to cover so much territory in so few pages, the text tends to be more factual than discursive. The graphs are beautiful in their simplicity. However, the documentation on which some of the charts are based is not always clear, and apparently the author has taken average values from certain references in literature plus his own experiences. This, of course, may not always be valid. There are some chapters which would perhaps evoke some disagreement; notably the chapter on the anemias which does not discuss the knowledge made available by the use of parenteral iron, and the chapter on x-ray pelvimetry which may have to be revised in the light of increasing known hazards of irradiation of the pregnant uterus.

The book is well written, concise, and shows the results of careful editing. I think it succeeds in the task set for it, and I think it can be recommended without reservation for the intern, the resident, and the busy general practitioner.

Erick Y. Hakanson, M.D.



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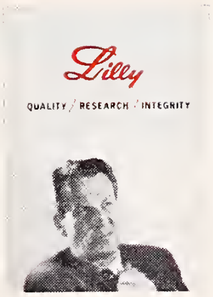

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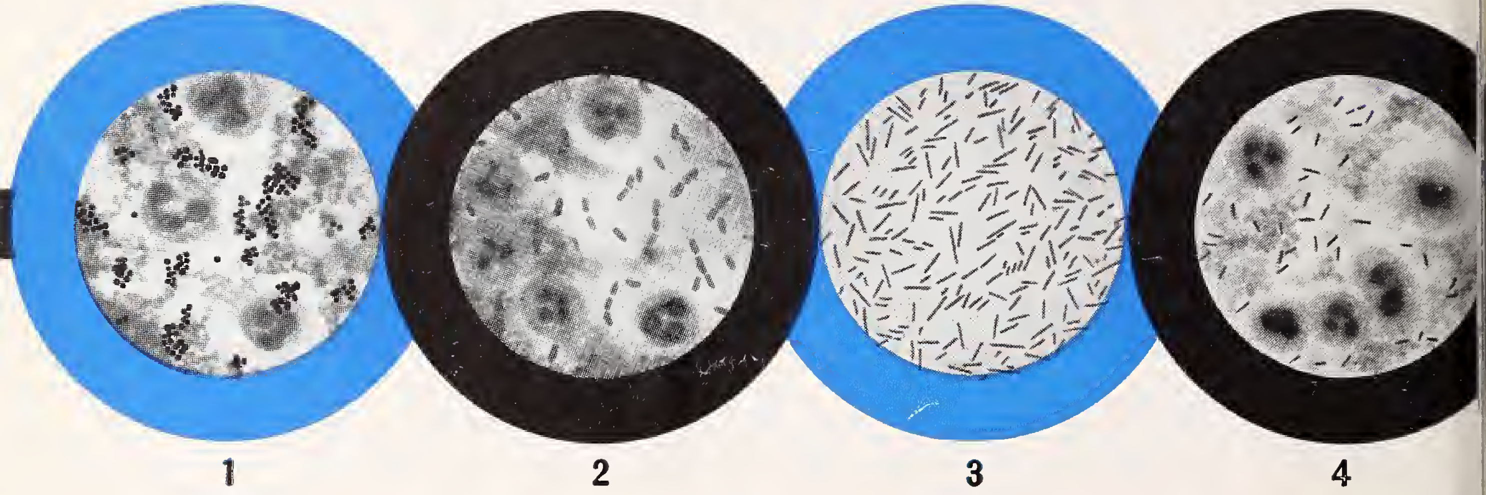

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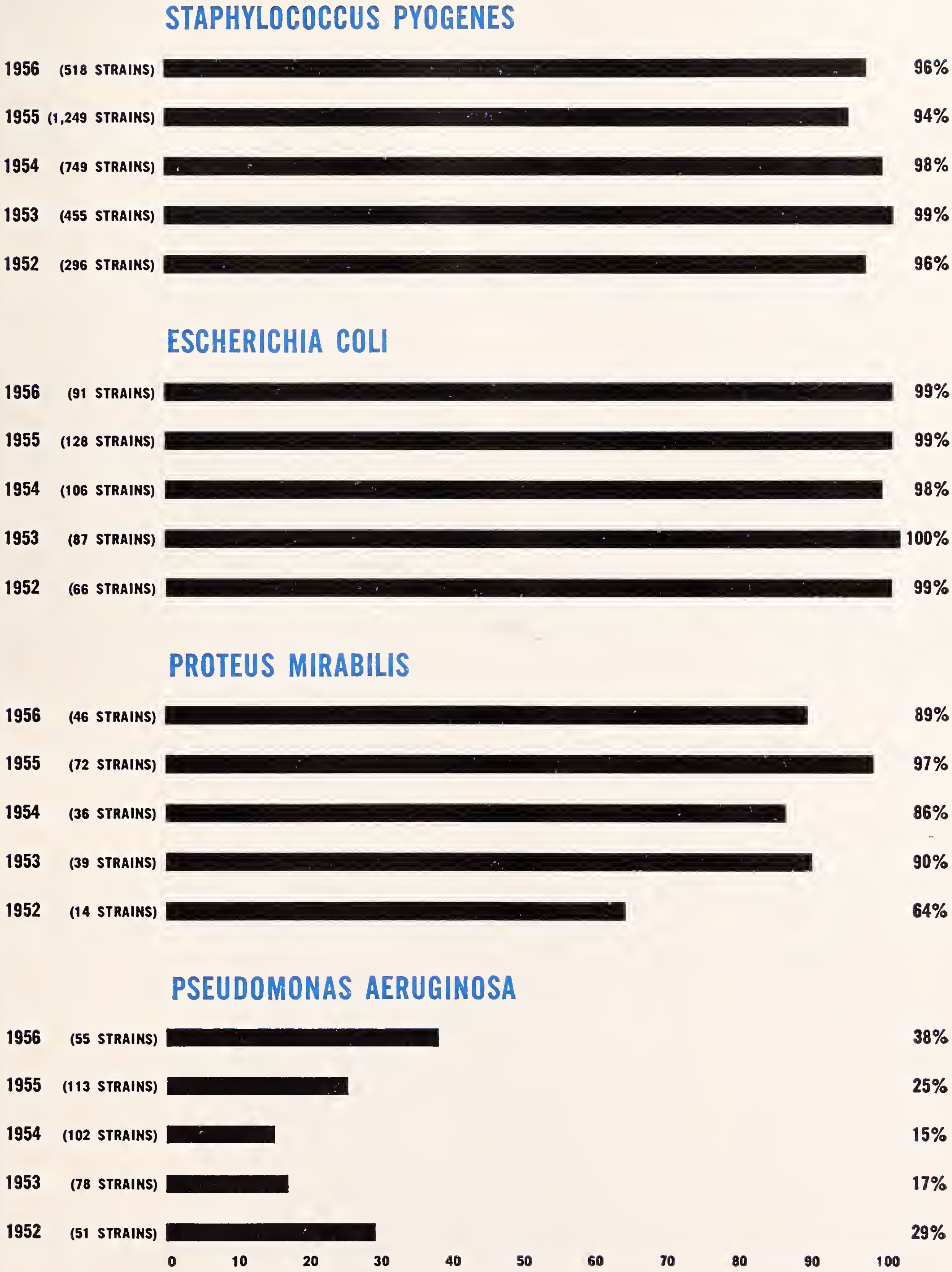

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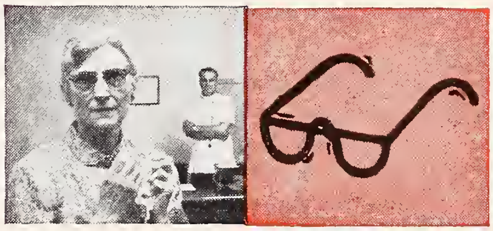

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Original Contributions

Clinical Aspects of Pheochromocytoma

WALTER F. KVALE, M.D.
Rochester, Minnesota

THE DIAGNOSIS of functioning pheochromocytoma is becoming less difficult as experience increases. Careful appraisal of the history, close scrutiny of the patient's age, weight and body build, and study of certain laboratory data often afford a clue at the first or second interview.

Since the first pharmacologic test for pheochromocytoma was introduced, my associates and I have had the opportunity of studying carefully fifty-seven patients who had pheochromocytomas. It is the purpose of this paper to present pertinent data on the diagnostic aspects and the results in fifty of these patients, to demonstrate that the diagnosis can be made if the possibility of this condition is kept in mind, to summarize indications for performing pharmacologic and other tests for pheochromocytoma, and to show that the transabdominal approach should be used and that cure is usually but not always possible.

Classification

The grouping of patients with hypertension caused by pheochromocytoma in those who have paroxysmal hypertension and those who have sustained hypertension is largely arbitrary. In general, those patients whose tumors secrete epinephrine and norepinephrine intermittently have paroxysmal hypertension, whereas those whose tumors secrete pressor substances continuously have persistent hypertension. However, the latter group also may have attacks during which the blood pressure is even higher than usual.

A total of twenty-six patients were classed as having paroxysmal hypertension. All twenty-six complained of episodes associated with pronounced increase in blood pressure. Between these attacks, the blood pressure was normal. The remaining twenty-four patients were considered to have persistent hypertension. Although their blood pressure was increased almost continuously, in some it would be normal or approach normal at times. The periods of increased blood pressure sometimes were associated with "spells," although not all patients had them.

Symptoms

Paroxysmal Hypertension.—As already noted, all the patients who had paroxysmal hypertension complained of spells, or attacks. Headache of varying intensity, but usually extremely severe, was the commonest symptom and was present in all except one patient. Sweating and palpitation were the next commonest symptoms. Also present were tachycardia, great anxiety, nervousness, pallor or flushing of the face, nausea and vomiting, pain in the thorax and abdomen, pain and numbness in the legs, and tingling and coldness of the hands and feet. In any patient, one or more of these symptoms could be lacking.

The duration of the illness varied from six weeks to ten years, the usual time being two to five years. The length of the history had no bearing on the secondary vascular damage that the intermittent discharge of the pressor amines might have produced. One woman who had been having bouts of excruciating headaches as frequently as several times a week for ten years had normal ocular fundi and a normal vascular system. On the other hand, a thirty-eight-year-old man who had had symptoms for only eight months had both coronary occlusion and thrombosis of the basilar artery before the diagnosis could be made.

The frequency of the attacks varied from as often as ten to twenty-five a day to once a day or even to only once every two to three months. They usually lasted only ten to fifteen minutes, but some persisted only a few seconds and others

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for several hours or days. Weakness and exhaustion commonly followed an attack. The usual story was that the attacks increased in frequency but not in severity. Between attacks, the patients were generally in good health.

Persistent Hypertension.—The twenty-four patients classed as having persistent hypertension had more or less continuous hypertension. However, ten complained of attacks similar to those experienced by patients who had only paroxysmal hypertension. During these attacks, the blood pressure increased to even higher levels. The usual picture was that of increasingly severe headaches, excessive perspiration, nervousness, palpitation and loss of weight. All twenty-four patients with persistent hypertension were thin, although two patients seen recently were slightly obese. If the condition has been unrecognized, changes due to secondary vascular damage, such as loss of vision, coronary occlusion, cerebrovascular thrombosis and congestive heart failure, may be responsible for the chief complaints.

Symptoms had been present for three weeks to sixteen years, the average being three to four years. The duration of the symptoms closely paralleled the duration of the hypertension, although hypertension in one case was not discovered for five years after the onset of symptoms.

Findings at Examination

Paroxysmal Hypertension.—The average age of patients with paroxysmal hypertension was forty-four years, ranging from twenty-six to fifty-nine. They tended to be older than those who had persistent hypertension. Only one patient was in the third decade of life; most (ten) were between thirty and forty years of age.

The height ranged from 62 to 72 inches (157 to 182 cm.). The average height of twenty-one patients was 65 inches (165 cm.). The patients also tended to be thin, ranging in weight from 106 to 192 pounds (48 to 87 kg.), with an average of 134 pounds (61 kg.). The patient who weighed 192 pounds was 70 inches (178 cm.) in height.

The level of blood sugar during a period of fasting was determined in twenty-five of these twenty-six patients under normotensive conditions; values greater than normal were found in eleven. The basal metabolic rate also was determined in twenty-five of the twenty-six patients. Values were more than +20 per cent in five, the highest rate being +33 per cent; they were between +11 and +20 per cent in five patients and were normal in fifteen. The basal metabolic rate was of aid in making the diagnosis of pheochromocytoma in only six cases. In general, the basal metabolic rate and level of blood sugar were not of material help in making the diagnosis in this group.

Excretory urograms were made in all cases. They gave evidence of a mass above one of the kidneys or displacement of the kidney in seven of the twenty-six cases.

Aortograms, perirenal injections of air and presacral injections of oxygen have not been used by us because localization is not considered necessary before operation. These procedures also entail an unnecessary risk to the patient.

Persistent Hypertension.—The average age of the twenty-four patients was thirty-five and six-tenths years, ranging from twelve to sixty-seven years. However, sixteen of the twenty-four were less than forty years of age and only two were more than sixty, being sixty-seven and sixty-five years old, respectively.

The average height of twenty of these twenty-four patients was 65 inches (165 cm.). The average weight of twenty-three of them was 122 pounds (55 kg.). Only five of the patients weighed more than 134 pounds (61 kg.). The two heaviest, weighing 169 and 160 pounds (77 and 73 kg.), were both 70 inches (178 cm.) tall. Eleven weighed 113 pounds (51 kg.) or less. The range in weight was 90 to 169 pounds (41 to 77 kg.). These patients generally were much thinner than those who had paroxysmal hypertension.

The basal metabolic rate was determined in twenty-one of the twenty-four patients. It ranged from +8 to +101 per cent and was more than +20 per cent in sixteen. Hypermetabolism without hyperthyroidism was of significant help in making the diagnosis in eighteen of these patients. In only three patients was the basal metabolic rate less than +10 per cent.

The level of blood sugar was determined in seventeen of the twenty-four patients; it ranged from 107 to 256 mg. per 100 ml., and in ten patients it was 120 mg. or more.

The excretory urogram was of help in localizing the tumor in four of the twenty-four patients. In one instance, however, the urogram indicated that the tumor might be on the right, whereas actually it was found on the left.

Findings at Operation

Paroxysmal Hypertension.—At the time of operation, nineteen tumors were removed from the right adrenal and five from the left. The tumors were bilateral in the two remaining patients. In one case of bilateral lesions, two tumors were removed from the right adrenal and one from the left; in the other case of bilateral tumors, the mass on the right was removed but the one on the left was not.

Seven of these twenty-six patients had gallstones. These were found before, at the time of, or after removal of the pheochromocytoma. The large percentage of gallstones in this series of cases suggests more than coincidence.

Persistent Hypertension.—There were thirteen tumors in the right adrenal, thirteen in the left and a total of six extra-adrenal tumors, three of which were inoperable metastatic lesions at the time of operation. One of the remaining extra-adrenal tumors was retroperitoneal on the left side, and one was situated behind the structures at the hilus of the liver. In one patient who had multiple tumors, three lesions arose from the right adrenal, one arose from the left adrenal and an additional tumor was found behind the right lobe of the liver. Thus, a total of thirty-two tumors were found in these twenty-four patients. None of them had gallstones. In one other case, two more tumors were found along the aortic chain at re-operation.

Type of Tumor and Results

Paroxysmal Hypertension.—In the twenty-six patients who had paroxysmal hypertension, all tumors except one were benign, and this one was considered benign until it recurred. Re-operation disclosed a fixed mass in the right adrenal region with metastasis to the liver.

Follow-up information was obtained on twenty-four of the twenty-six patients. All except two were living at the last report. One of the two who died had active pulmonary tuberculosis at the time of operation and subsequently died of this disease; the other died from causes other than pheochromocytoma ten years after the tumor was removed. Five patients have had further difficulty; one had residual effects from a cerebral infarct, one had metastasis found at re-operation and treated roentgenologically, and the other three had only minor difficulties. The remaining seventeen patients were living and well at last report.

Persistent Hypertension.—The lesions in seventeen of these twenty-four patients were benign; one patient had a malignant tumor and three had tumors that were apparently benign on pathologic examination but metastasis occurred later. Inoperable fixed pheochromocytomas were found at operation in the remaining three patients, all of whom died soon afterward of metastasis.

Thus, seven of these twenty-four patients with persistent hypertension had potentially fatal pheochromocytomas, and six of these have died from the effects of malignancy. The seventh has had recurrent tumors removed but was still living, although he had metastasis. One other patient died, presumably from metastasis from a carcinoma of the pancreas, and still another one died after sympathectomy.

The remaining fifteen patients were living and well at last report, although one had residual homonymous hemianopsia that developed after operation. Three have noted residual mild hypertension. In all patients in whom the changes in the retinal vessels were those of hypertension Group 3 or 4, the changes have reverted to those of hypertension Group 1 or 2.

Surgical Aspects

Frequent determination of blood pressure and its proper control are among the most important procedures during operation on a patient who has pheochromocytoma. Characteristically, the blood pressure fluctuates widely during removal of a pheochromocytoma. It usually increases sharply with induction of anesthesia. This increase commonly continues as the incision is made and becomes more pronounced as the tumor is palpated and mobilized. After the tumor is removed, the blood pressure decreases sharply to levels typical of shock. This drop is usually most pronounced in patients who had persistent hypertension before operation. Actually, if this decrease does not occur at this time, the presence of an additional tumor that has not been removed should be suspected. Because of these wide fluctuations in blood pressure, which may have serious results if not properly treated, it is essential to have depressor as well as pressor agents readily available during operation.

It is our practice always to insert a needle in a vein and start a slow infusion of a 5 per cent solution of dextrose before induction of anesthesia. Phentolamine (regitine) hydrochloride in doses of 5 mg. is used as the depressor agent, and levarterenol (levophed) bitartrate in doses of 4 mg. is used as the pressor agent. When the blood pressure increases with induction of anesthesia, it may be advisable to inject regitine. This is always true as the tumor is palpated and mobilized. After removal of the tumor, levophed is employed. If the blood pressure does not decrease after removal of a tumor, further confirmation of another tumor may be obtained by administration of regitine, which, under these circumstances, may be expected to cause a definite decrease in blood pressure.

Various surgical approaches have been used in operations for pheochromocytoma. It is our belief that a transverse upper abdominal incision is the approach of choice. This exposure permits simultaneous exploration of both adrenal glands through a single incision and also affords opportunity for general abdominal exploration. It obviates the need for examinations for localization of a tumor in one adrenal gland, such as perirenal or presacral injection of air or aortography, since both adrenal glands are routinely palpated and inspected at operation. Exploration of the abdomen and opposite adrenal gland from a posterolumbar incision on one side has not been satisfactory in our experience.

Although it is true that pheochromocytoma usually is a single benign tumor in one adrenal gland, these tumors may be multiple on one side or bilateral, they may be malignant, and they may be located wherever chromaffin tissue is found. Because of the possibility of multiple, bilateral or ectopically located tumors, both adrenal glands should be explored in every patient and, in addition, general abdominal exploration should be done. Particular attention is given to the region of the great vessels in the abdomen and the base of the mesentery of the small intestine. Fortunately, almost every patient who has a pheochromocytoma is thin, which facilitates the abdominal approach.

Appropriate postoperative care after removal of a pheochromocytoma is extremely important. This is likely to be especially true if the patient has had sustained hypertension before operation. The main concern during the postoperative period is maintenance of adequate blood pressure. The pronounced hypotension that becomes evident on removal of a functioning pheochromocytoma may persist for hours or several days after operation unless proper supportive measures are employed. These patients should be kept under constant observation, with frequent determinations of blood pressure until reasonable levels of blood pressure persist without support. It is our practice during this period of pronounced hypotension to use a slow but continuous drip of 1000 ml. of a 5 per cent solution of dextrose to which 4 mg. of levophed has been added.

Indications for Tests for Pheochromocytoma

One of the problems in diagnosis is to ascertain under what conditions the presence of a pheochromocytoma should be suspected and when laboratory tests for pheochromocytoma should be ordered. Our data indicate that patients of the following types should be scrutinized with the possibility of pheochromocytoma in mind; if other diagnostic possibilities are ruled out, it might be wise to perform pharmacologic tests for pheochromocytoma on them:

1. Any patient who complains of "spells" associated with headache, perspiration, thoracic and abdominal pain, nervousness and vasomotor phenomena should have a pharmacologic test.

2. Thin patients with histories of fluctuating hypertension but whose blood pressure is within normal limits on examination probably should have a histamine test. All other thin patients with hypertension should undergo the appropriate test.

3. All young persons with hypertension should be tested. Only three of our twenty-four patients with persistently functioning pheochromocytomas were more than fifty years old, and sixteen were less than forty years of age. If results of regitine tests in such persons are negative or equivocal, the procedure should be repeated, other tests should be performed, or the concentration of pressor amines should be determined.

4. All persons who have hypermetabolism without hyperthyroidism should be investigated. In sixteen of our twenty-four patients with persistently functioning pheochromocytomas, the basal metabolic rate was more than +20 per cent and hyperthyroidism and other causes of hypermetabolism had been excluded.

5. All persons with a short history of hypertension should be considered for tests. Hyper-

tension had been known for only three weeks in one of our patients, an eighteen-year-old girl. In two patients, hypertension was discovered on examination at the clinic. However, the symptoms of hypertension ordinarily closely approximate the known duration of the hypertension.

6. All patients who have severe hypertension Group 2, 3 or 4 who are not included in the foregoing categories should undergo tests. Fifteen of the twenty-four patients with persistently functioning pheochromocytomas had ocular evidence of hypertension Group 3 or 4.

7. Patients who respond in a paradoxic fashion to the ganglion-blocking agents should be considered for pharmacologic tests. In one of our patients, severe increase in blood pressure occurred after the initial hypotensive reaction from subcutaneously injected hexamethonium.

8. When a patient responds unsatisfactorily to anesthesia and the blood pressure increases, the administration of the anesthetic agent should be stopped immediately, for this reaction could indicate a pheochromocytoma.

Summary and Conclusions

A study has been made at the Mayo Clinic on fifty patients who had surgically proved pheochromocytomas. These tumors, although rare, can be detected in many instances by careful appraisal of the history, close scrutiny of the patient's age and weight, and use of either the histamine or phentolamine (regitine) test or both. If any question remains, determination of the pressor amines in the blood during the time of the highest blood pressure is needed. Pheochromocytomas appear to be peculiar to thin people, especially those who have persistent hypertension. Those patients with hypertension who are young, are thin, have lost weight and have hypermetabolism without hyperthyroidism should have the pharmacologic tests for this lesion.

The tumors are usually benign. Their removal may prevent secondary vascular changes, such as visual impairment, coronary occlusion, congestive heart failure and cerebrovascular accidents, and may be lifesaving. The tumors may be malignant and some that at first are considered benign later may become malignant and metastasize. They may recur. Therefore, the diagnosis and removal of pheochromocytomas do not always insure a promising outcome. The pheochromocytomas in eight of these fifty cases (16 per cent) were malignant or have become malignant; the largest number of malignant lesions occurred in persistent hypertension. More than a coincidental relationship apparently exists between gallstones and pheochromocytoma.

Pheochromocytomas may be located anywhere in the abdominal cavity. It is not necessary to localize them preoperatively, since an anterior abdominal approach allows free exploration. After removal of a pheochromocytoma, patients should be examined frequently to be sure that recurrence has not developed nor metastasis occurred. If either is present, further operative or roentgenologic treatment may prolong the life of the patient.

EMERGENCY IDENTIFICATION CARD FOR PATIENTS ON ANTICOAGULANT THERAPY

An "emergency" identification card for the protection of patients on long-term anticoagulant therapy is now available to physicians from the American Heart Association and its affiliates.

The card, designed as a wallet insert, was developed as a result of requests from physicians seeking to protect their patients on anticoagulants in case of accident, dental surgery or other treatment that may induce bleeding. It points out that the bearer "is being treated with anticoagulants which slow down clotting of the blood." In case of emergency—bleeding, injury or illness—the card advises that a doctor be called, since the patient may require an antidote.

The card contains space for the name, address and phone number of the individual's physician. There is also space to indicate the kind of anticoagulant prescribed and the patient's blood type. The card was designed with the approval of the Committee on Prothrombin Determinations of the American Heart Association.

In addition to making the anticoagulant identification card available to physicians, the Heart Association also is calling it to the attention of dentists, hospital emergency room personnel, nurses, police and others who most commonly handle emergencies.

Physicians may obtain samples of the identification card from their local Heart Association or from the American Heart Association at 44 East 23rd Street, New York City.

Pressor Amines in Pheochromocytoma

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THE FLUOROMETRIC method of Weil-Malherbe and Bone,[1] with some modifications,[2] was used for the quantitation of epinephrine and norepinephrine in the plasma of patients with essential hypertension and those with pheochromocytomas.

In healthy men and women, the highest concentration of epinephrine-like substance per liter of plasma was 3.6 micrograms. The concentration of epinephrine-like substance in more than 90 per cent of an equally large number of patients with essential hypertension was less than 3.6 micrograms per liter of plasma; however, several of these patients occasionally had concentrations of 4 to 5 micrograms.

In twenty-three patients with pheochromocytomas, all but one of those who had sustained hypertension and two of those who had paroxysmal hypertension had concentrations of epinephrine-like substance greater than 3.5 micrograms per liter of plasma; values for the sum of epinephrine and norepinephrine when hypertension was sustained or when blood was drawn during a paroxysmal episode of hypertension were more than 8.8 micrograms in these same patients. Further studies on the three patients with pheochromocytomas and normal plasma concentrations of pressor amines showed that the amount of pressor amines increased above normal in the patient with persistent hypertension and in one of the two patients with paroxysmal hypertension after a provocative histamine test. Unfortunately, the effect of histamine was not studied in the third patient, but the plasma concentration of pressor amines was abnormally increased during anesthesia in this instance. Administration of a general anesthetic agent often simulates a provocative test in patients who have pheochromocytoma.

Patients with renal insufficiency should be carefully evaluated, since this condition may be associated with retention of fluorescent substances other than epinephrine and norepinephrine that give falsely high values for plasma pressor amines. Some patients with brain tumors or lymphomas have concentrations of plasma pressor amines that are above the range of normal. It is uncertain whether the substances measured as pressor amines in patients who have lymphomas are epinephrine or norepinephrine or both or some other substances. One patient with acute pancreatitis had greatly increased values for epinephrine-like substance in the plasma; however, the presence of hyperbilirubinemia in this patient made it uncertain whether an actual increase in plasma pressor amines was present. My associates and I have found that bile pigments or hemolysis will interfere with the fluorometric quantitation of epinephrine and norepinephrine in plasma by producing sufficient fluorescence to indicate erroneously increased concentrations of pressor amines.

We recently observed a patient with persistent hypertension who for many years had been using "asthma-nefrin," a solution of epinephrine hydrochloride, in a nebulizer for treatment of recurrent attacks of asthma. Mild azotemia also was present. Despite the fact that asthma-nefrin had not been used for about forty hours, the concentration of epinephrine-like substance was 5.1 micrograms per liter of plasma and results of the phentolamine (regitine) test were equivocal. The patient subsequently used nebulized asthma-nefrin during an acute attack of asthma; forty-one hours later, the concentration of epinephrine-like substance averaged 12.2 micrograms and results of the regitine test again were equivocal. Because a diagnosis of pheochromocytoma could not be excluded, exploratory laparotomy was performed but a tumor

(Continued on Page 321)

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Laboratory Diagnosis of Pheochromocytoma

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IN OTHER presentations in this symposium, Dr. Kvale and Dr. Manger have discussed certain aspects of the diagnosis of pheochromocytoma. Despite the excellent method described by Dr. Manger, which is sensitive and extremely useful, the diagnosis of pheochromocytoma is still a challenge and, if anything, is becoming more difficult to make. Some additional laboratory methods for this diagnosis, with their merits and defects, also should be considered.

Pharmacologic Tests

The patient who has a pheochromocytoma will have either paroxysmal or sustained hypertension. When the hypertension is paroxysmal, the most effective drug in my experience for the diagnosis of pheochromocytoma is histamine, which stimulates discharge of pressor substances from the tumor and produces attacks similar to those occurring spontaneously. Likewise, if the patient has mild hypertension and a history of paroxysmal attacks, both histamine and phentolamine (regitine) may be used. When the patient has sustained hypertension, regitine alone is used, because it decreases blood pressure by blocking the pressor effect of epinephrine and norepinephrine in the blood if a pheochromocytoma is present.

Technique.—The pharmacologic tests are simple, and a clinician may do them in his office. First, with the patient lying comfortably, the blood pressure is observed for half an hour to obtain the correct basal value. This is highly important in the interpretation of the results after use of histamine or regitine.

Histamine Test.—As a preliminary to the histamine test, a cold pressor test is carried out by immersing one of the patient's hands well above the wrist in a pail (8 inches in height) containing water at 4° C., keeping it immersed for one minute and measuring the blood pressure in the opposite arm at fifteen, thirty and sixty seconds while the hand is in the water. The highest reading during this stimulus represents the lability of the blood pressure. If a patient has a history of having high blood pressure, such as 230 mm. of mercury systolic and 160 mm. diastolic, at one time and at another time a low blood pressure of perhaps 130/90, the blood pressure under basal conditions may be 130/90 and then increase to 230/160 while the hand is immersed in the cold water. Such a patient has an extremely labile blood pressure and in most instances will not have a pheochromocytoma. However, if the increase of blood pressure during the cold pressor test is much less, pheochromocytoma may be suspected.

This cold pressor test is an integral part of the histamine test, because it should be used as the measuring stick for the response of the blood pressure two minutes after an intravenous injection of histamine. A cold pressor test is not done when the diastolic blood pressure is 150 mm. of mercury or more.

Histamine is given only when the basal blood pressure is less than 170/110. The histamine test as done at the Mayo Clinic is as follows: 0.05 mg. of histamine base in 0.5 ml. of isotonic solution of sodium chloride is injected intravenously. Determinations of the blood pressure are made every thirty seconds for the next two minutes. During this time, the blood pressure always decreases thirty seconds after the injection of histamine if the material has entered the vein. Immediately thereafter, the blood pressure increases rapidly and usually reaches the maximum in two minutes. As soon as the histamine is injected, the needle is left in the vein, the empty syringe is removed and a syringe containing regitine is attached. If a pheochromocytoma is present, the

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characteristic clinical signs and symptoms of a severe episode appear concomitantly with the increase in blood pressure; the increase in systolic pressure may range from 30 to 104 mm. of mercury above basal pressure, and that in diastolic pressure may be from 25 to 56 mm. If the increase in blood pressure is caused by release of pressor amines, the regitine can be injected at any time when the blood pressure becomes alarmingly high; within one minute this drug produces complete cessation of the clinical signs and symptoms and a sudden rapid decrease in the blood pressure. Thus, histamine can be given safely and two positive reactions will be noted. If the patient has only mild essential hypertension, the blood pressure will not increase so much after the injection of histamine, and the headache will not disappear nor will the blood pressure decrease rapidly after the injection of regitine. If regitine is given alone to a patient with labile hypertension between paroxysms, the blood pressure may not change in the presence of a pheochromocytoma because pressor amines are absent in the blood between attacks. Therefore, falsely negative results will be obtained.

Regitine Test.—For the patient who has sustained hypertension (a basal blood pressure above 170/110), 5 mg. of regitine is administered intravenously; in the presence of pheochromocytoma, the blood pressure should decrease at least 35 mm. of mercury systolic and 25 mm. diastolic in 4 minutes and then increase to approximately the previous basal level in 10 to 15 minutes. Regitine has a tendency in some patients to produce some decrease in blood pressure during the first 1 or 2 minutes in the absence of a pheochromocytoma, but the blood pressure then returns to a level that is considered negative for such tumors.

Precautions.—The question often arises as to what precautions may be necessary to obtain correct results in these pharmacologic tests. One difficulty may be a difference in the blood pressure of the two arms, which may give falsely positive results with either histamine or regitine. If the blood pressure in both arms is measured simultaneously, a truly negative reaction to histamine and regitine then will become apparent. On the other hand, if a pheochromocytoma is present, the positive response will be evidenced in both arms. Therefore, the blood pressure is measured routinely in both arms of all patients; if any disparity occurs, the blood pressure then is determined simultaneously in both arms during pharmacologic tests.

A second precaution concerns previous medication. Before any of these tests, administration of any sedative or narcotic should be prohibited for at least forty-eight hours and possible self-medication should be checked or the pharmacologic tests may yield falsely positive results. In patients who have paroxysmal hypertension, sedatives inhibit the increase of blood pressure during the cold pressor test that is the measuring stick. As a result, the increase of blood pressure during the histamine test may be greater than that during the cold pressor test, thus indicating the presence of a tumor that does not exist. In patients who have sustained hypertension, the administration of potassium thiocyanate, barbiturates, meperidine (demerol), morphine, chloral hydrate and probably many other sedatives may cause a decrease in blood pressure typical of that produced in the presence of pheochromocytoma after the intravenous administration of regitine. On the other hand, various antihypertensive drugs may produce falsely negative results in the pharmacologic tests in patients with pheochromocytoma. Since most of these drugs act longer than do the sedatives, the difficulties are even greater. I have observed three patients who were receiving antihypertensive drugs in whom the results of the regitine test were initially negative but later reversed to positive after use of these drugs was discontinued and in whom tumors were subsequently found at operation. The ideal time to do these tests is before any drugs are given. If this is impossible, the administration of antihypertensive drugs should be discontinued for ten to twelve days. Thus, if previous medication has not been used or if use of the drugs has been stopped for an adequate period, the histamine test yields clear-cut positive or negative results for patients who have paroxysmal hypertension and the regitine test gives unequivocal results for patients who have sustained hypertension. Furthermore, these results may be obtained in an hour, and the tests may be carried out by the clinician. More than one test always should be done before an operation is performed.

Chemical Tests: Pressor Amines

The chemical tests for pheochromocytoma discussed by Dr. Manger present pitfalls, just as do the pharmacologic tests, because some pheochromocytomas may not secrete continuously. This is true in patients with paroxysmal hypertension and probably in some patients with sustained hypertension who have pheochromocytoma. Blood or urine collected when the tumor is not secreting will contain small or normal amounts of epinephrine and norepinephrine, which indicates no tumor. However, if the blood is obtained before and at the maximal increase of blood pressure during a histamine test, the pressor amines in the blood will be greatly increased in a patient with pheochromocytoma and a correct diagnosis can be made, as positive results in three tests then will be available (two pharmacologic procedures and one chemical). On the other hand, no significant increase of epinephrine or norepinephrine in the blood is noted after the intravenous injection of histamine in patients without pheochromocytoma.

Negative results may be obtained with the chemical test for urinary pressor amines in the presence of pheochromocytoma. One patient had exhibited negative results in several tests for urinary catecholamines done elsewhere. A regitine test at the clinic gave positive results for pheochromocytoma, and a significant increase of pressor amines was noted in the blood. An 80-gm. pheochromocytoma was found at operation.

In contrast, in certain conditions such as azotemia, jaundice and lymphoblastoma, fluorescent substances other than pressor amines are present in the blood and appear as increased amounts of "pressor amines" in the chemical test. In one patient with lymphoblastoma, the pharmacologic tests for pheochromocytoma gave negative results but the clinical findings and an increase of "pressor amines" indicated tumor. The patient was explored and later died; pheochromocytoma was not found at necropsy.

Patients receiving chlorpromazine (thorazine) may have increased levels of fluorescent substances other than pressor amines; one such patient with a high level of substances presumed to be pressor amines died and an adrenal tumor was not found. Tetracycline (achromycin) apparently causes the appearance of fluorescent substances other than pressor amines; extremely high levels (7.4 to 40 micrograms) of "pressor amines" were found in four patients receiving this antibiotic. After use of the drug was stopped for four to six days, decreased levels of "pressor amines" were present. One of these patients was explored and adrenal tumors were not found. It was significant that the regitine test gave negative results in these patients. A normal volunteer (Dr. Forester) took three doses of 250 mg. each of achromycin orally six hours apart. After the third dose, the "pressor amines" had increased from 1.9 to 6.9 micrograms. Other drugs also may have such fluorescent properties.

Two further difficulties in the measurement of pressor amines have arisen with the use of antihypertensive drugs. First, while pressor amines may be increased in patients with pheochromocytoma who are receiving antihypertensive drugs, evidence exists that these agents may depress the amount of pressor amines during their initial administration. However, growth of the tumor is not inhibited and, as it grows, the increase in blood pressure may be re-established, as well as an increase in the amount of pressor amines. Secondly, an annoying side reaction after the use of antihypertensive drugs is nasal stuffiness. Many vasoconstrictor substances are used locally to alleviate this difficulty. These substances produce high levels of pressor amines in the blood and increased amounts of catecholamines in the urine; such increases have been interpreted as an indication of pheochromocytoma. These local vasoconstrictor drugs produce a profound decrease in blood pressure that may persist for as long as forty minutes after the intravenous injection of regitine. This is in contrast to the situation in a patient who has a pheochromocytoma; in this instance, the decrease in blood pressure may be as great but the pressure begins to increase in four to five minutes and often approximates the basal level within ten minutes. The prolonged lowering of blood pressure after injection of regitine alerts the physician to the possibility that something other than a pheochromocytoma is producing the response.

Comment

It is apparent that no single test is adequate in testing for pheochromocytoma. The sedatives produce falsely positive results with histamine and regitine, whereas antihypertensive drugs produce falsely negative results. However, by discontinuing use of sedatives for forty-eight hours and of antihypertensive drugs for a week or more, reliable results with regard to pheochromocytoma can be obtained.

Under these conditions, a correct diagnosis can be made by pharmacologic tests. A total of 11,706 pharmacologic tests have been carried out on 10,623 patients at the clinic with no untoward effects. Pheochromocytomas have been correctly diagnosed and found at operation in fifty-seven patients.

Use of only the chemical test for pressor amines may be unreliable in the diagnosis of pheochromocytoma after administration of chlorpromazine, achromycin and perhaps other drugs unknown as yet, in patients with azotemia or jaundice, and in certain diseases such as lymphoblastoma. The same holds true in patients receiving vasoconstrictor drugs, such as patients with asthma or vasomotor rhinitis, or patients having the afore-mentioned side reactions from antihypertensive drugs. For the most part, use of drugs that influence the pressor amines must be discontinued for at least six days to obtain reliable results.

Summary

Correct results in pharmacologic and chemical tests for pheochromocytoma may be obtained if certain precautions are observed. Accurate determination of the basal blood pressure before the tests is necessary, as is discontinuance of previous medication, such as use of sedatives, narcotics and various antihypertensive drugs.

A cold pressor test is an integral part of the histamine test for pheochromocytoma. Simultaneous determination of blood pressure in both arms during either a histamine or a phentolamine (regitine) test will produce accurate results if blood pressure differs in the two arms.

Blood and urine collected for the chemical measurement of pressor amines shortly after a spontaneous attack or after induction of an attack by histamine contain increased amounts of pressor amines, which indicates the presence of pheochromocytoma. However, chemical tests alone are unreliable after the administration of chlorpromazine and tetracycline (achromycin) in patients with azotemia, jaundice or lymphoblastoma, and in patients receiving vasoconstrictor drugs, such as those with asthma or vasomotor rhinitis or those having side reactions from antihypertensive drugs.

When measures are taken to eliminate the situations that interfere with correct results and when full consideration is taken of the inherent potential shortcomings in the procedures, the use of both pharmacologic and chemical tests is of great value in the diagnosis of pheochromocytoma.

METHOD FOR DETECTING HEART LEAKS

A method for detecting hidden leaks in the valve between the left chambers of the heart has been developed by scientists at the National Heart Institute in Bethesda, Maryland.*

The new method has been described in technical detail in *The Journal of Clinical Investigation* (January, 1958) by cardiologists Eugene Braunwald and George H. Welch of the Institute's Laboratory of Cardiovascular Physiology, and surgeon Andrew G. Morrow of its Clinic of Surgery.

To reveal the leak, these investigators raise the arterial blood pressure by injecting the artery-constricting hormone, norepinephrine, into the patient's blood stream at a carefully measured rate. Meanwhile, they observe and record the effects of this increase in blood pressure on the pressure inside the left atrium.

A leaking valve is revealed if the pressure rises grossly in this chamber in response to the rise produced in the arterial blood pressure. In a normal heart, the pressure rise in this chamber is slight because such gross changes in arterial blood pressure are largely excluded from the atrium by a tightly closing mitral valve.

A combined condition of "stenosis" and "insufficiency" commonly affects the same mitral valve. The new diagnostic method is expected to be most useful against the combined condition, where the murmurs and blood pressure abnormalities of insufficiency are often masked by those of stenosis.

Detecting hidden mitral insufficiency before operating for stenosis may be of vital importance, because the valve-splitting operation usually performed for stenosis may actually worsen the heart trouble if the valve also tends to leak.

Following successful tests in dogs, the method was tried in twenty patients in the Heart Institute Clinic of Surgery. Seven of these had pure mitral insufficiency, seven had pure stenosis, and the other six had the mixed condition.

In the thirteen patients with insufficiency, norepinephrine caused the pressure in the atrium to rise momentarily to an average of 47.8 per cent of the rise in the arterial blood pressure—in some patients it went as high as 125 per cent. In the patients with pure stenosis, the increase in atrial pressure averaged only 8.5 per cent of the arterial pressure increase. This difference was great enough to form the basis for a method for detecting mitral insufficiency in difficult cases.

To record blood pressures in the left atrium, a slender filament of plastic catheter tubing was placed in the heart via the mouth, throat and windpipe—a catheterization technique called the "transbronchial puncture," introduced into the U. S. by Dr. Morrow. Dr. Morrow sprays the back of the patient's throat with novocain and then makes a puncture with a hypodermic needle at the point where the windpipe forks. He then threads the catheter through the bore of the needle into the atrium, which lies just below the fork. The needle is then withdrawn, allowing the patient to relax in comparative comfort, with one end of the flexible hollow thread of catheter in his heart and the other end emerging from his mouth to be attached to recording and sampling devices.

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*The National Heart Institute is one of the seven National Institutes of Health that comprise the research arm of the U. S. Public Health Service.

The Treatment of Hypertensive Emergencies

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MARKED AND SUDDEN elevation in blood pressure, regardless of cause, may become a direct threat to the patient's life even though that elevation may be only a part of the clinical syndrome. Under these circumstances, reduction of the blood pressure may be the primary therapeutic objective.

Hypertensive emergencies require therapeutic agents that will reduce the blood pressure rapidly. The agents available for this purpose vary considerably in effectiveness, depending upon the etiology of the blood pressure elevation. For example, hydralazine is very dependable in the therapy of toxemia of pregnancy and acute glomerulonephritis. However, it is of little value in the treatment of so-called "malignant essential hypertension." Ganglionic blocking agents are useful for the treatment of an ambulatory patient with essential hypertension, but because they are primarily effective in the standing position, they are much less valuable for the therapy of the bedfast patient with a severe hypertensive crisis. Veratrum extracts, when administered parenterally, are effective in all types of hypertension (except pheochromocytoma), but due to the severity of the side effects, these compounds should be used only after the less potent agents have been tried. Thus, if these drugs are to be used to best advantage in the treatment of hypertensive emergencies of various etiologies, the therapist must be acquainted with the clinical pharmacology of the different agents. It is the purpose of this report to present a general approach to the treatment of hypertensive emergencies and to discuss some pertinent clinical pharmacodynamic problems.

Although the exact physiology underlying the majority of cases of hypertensive emergencies associated with essential hypertensive vascular disease is not known, the importance of the autonomic nervous system in its maintenance seems well-founded. It has been established that drugs that produce partial blockade at various points along the sympathetic pathways from the higher centers to the neuroeffector site will reduce the arterial blood pressure in these instances (Fig. 1), regardless of the degree of its elevation and without regard to the underlying cause of the hypertension.

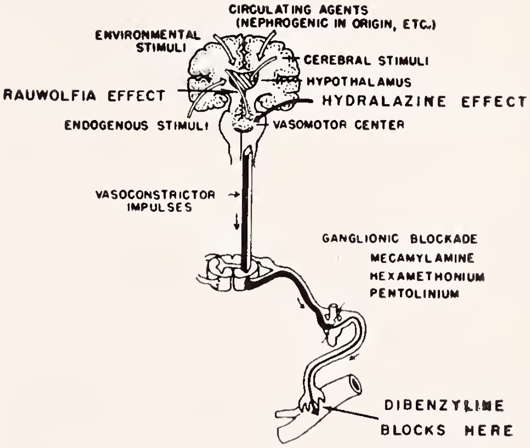

Fig. 1. Pharmacology of hypertension and antihypertensive agents used in the treatment of hypertensive emergencies. Rauwolfia and Hydralazine act centrally to depress vasoconstrictor impulses traveling from the brain to the blood vessels. Veratrum acts centrally as well as reflexively by stimulating afferent impulses in the heart and great vessels which are transmitted to the brain as vasodepressor reflexes. Following administration of the peripherally acting drugs, sympathetic impulses are blocked and the reduction in blood pressure is primarily in the orthostatic position.

The drugs used for the treatment of hypertensive emergencies may be classified on the basis of three sites of action within the sympathetic nervous system i.e., central, ganglionic, and adrenergic blocking agents:

(1) The most effective centrally-acting agents are extracts of Rauwolfia serpentina (reserpine rescinnamine and deserpidine), Veratrum preparations (cryptenamine, protoveratrine, alkavervir) and hydralazine (Apresoline) which suppress vaso-

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Presented at the Symposium on Peripheral Vascular Disease co-sponsored by the Minnesota Heart Association and the Mayo Foundation, Rochester, Minnesota, September 25, 1957.

TABLE I. BLOOD PRESSURE AND SYMPTOMATIC RESPONSE

Continuous Infusions of Intravenous Veriloid for the Treatment of Severe Encephalopathy

Patient	Age	Known Duration (yr.)	Diagnosis	Pulse Rate		Blood Pressure			Side Reactions	Infusion Period (hr.)	Symptomatic Improvement (Encephalopathy)
				Control	During Infusion of Veriloid	Control Mean	During Veriloid Infusion				
							Mean	Range			
1	42	10	CN*	77	50	210/140	156/104	180/120-130/ 80	None	72	++
2	24	8	HCVD†	66	44	260/150	155/ 90	170/100-130/ 72	None	48	++
3	36	7	HCVD	82	80	300/210	180/125	200/140-150/110	Weakness	96	+++
4	51	2	HCVD	100	60	290/160	140/120	160/130-130/108	Hiccough, vomiting	24	+
5	28	13	CN	107	108	224/162	160/110	196/140-110/ 80	Restless	48	+++
6	48	1	HCVD	120	100	230/164	150/ 90	160/108-138/ 80	Dizzy, weak, nausea	48	+++
7	52	10	HCVD	80	64	248/130	162/ 90	180/104-148/ 82	Dizzy	48	+++
8	33	6	CN	90	70	190/120	110/ 66	130/100- 90/ 58	Vomiting	24	++
9	29	6	CN	80	64	220/160	130/ 95	150/108-110/ 90	None	18	Pt. died
10	27	4	HCVD	104	70	265/170	155/105	170/115-130/100	Vomiting	72	+++
11	60	7	HCVD	84	70	235/150	170/110	190/120-135/ 90	Nausea	72	+++
12	55	0.5	CN	100	50	225/145	180/130	200/140-170/120	None	96	+
13	48	0.3	HCVD	72	58	310/220	240/160	260/180-200/130	Weakness, hiccough	48	0
14	49	1	HCVD	84	76	200/140	130/100	158/115-120/ 80	Vomiting	24	0
15	53	2	HCVD	80	86	190/125	130/110	170/120-110/ 98	Hiccough, nausea	72	++
16	56	1	HCVD	76	72	240/162	150/100	170/110-140/ 96	None	96	0
17	54	1	HCVD	90	56	300/210	158/110	200/130-110/ 90	Hiccough, weakness	24	+++
Mean	44	4.7		88	69‡	243/159	156‡/107‡			55	

*CN—Chronic nephritis.
†HCVD—Hypertensive cardiovascular disease.
‡Statistically significant change from control values ($p<0.01$).
Courtesy, Moyer, et al: The American Journal of Medicine, 14:175, 1953.

TABLE II. COMPARISON OF CEREBRAL HEMODYNAMIC RESPONSE TO ACUTE BLOOD PRESSURE REDUCTION

With Various Agents in Hypertensive and Normotensive Subjects: Mean Values

Type of Patient	Drug Administered	Mean Blood Pressure mm. Hg.		Cerebral Blood Flow cc/min.		Cerebral Oxygen Consumption cc/min.		Cerebrovascular Resistance		Arterial P CO_2		Number Patients Studied
		C	D	C	D	C	D	C	D	C	D	
Severe hypertensives	Phenoxybenzamine (dibenzyline)	149	82	48	42	3.6	3.1	3.2	2.2	40	40	7
Severe hypertensives	Alkavervir (veriloid)	173	108	57	50	2.9	3.4	3.2	2.3	39	37	10
Severe hypertensives	Dihydroergocornine	160	122	58	58	3.5	3.6	2.9	2.0	38	39	12
Normotensives	Hexamethonium	99	62	57	42	3.2	2.9	1.8	1.5	41	40	8
Normotensives	Dihydroergocornine	94	78	60	55	3.8	3.5	1.7	1.5	40	40	5

C=Control.
D=After drug.
Courtesy Moyer et al: American Journal of Medical Sciences, 228:563, 1954.

constrictor impulses at their sites of origin at or above the vasoregulatory centers.

(2) Ganglionic blocking agents, that is, hexamethonium (Methium), pentolinium (Ansolysen) chlorisondamine (Ecolid), mecamylamine (Inversine) and trimethaphan camphorsulfonate (Arfonad), act not only at sympathetic ganglia to block vasoconstrictor impulses but also at the parasympathetic ganglia to produce a variety of undesirable effects.

(3) The third major group of autonomic blocking agents are those that produce adrenergic blockade, such as phenoxybenzamine (Dibenzyline), at the neuroeffector site.

Pharmacodynamics

Cerebral function is improved by lowering the blood pressure in hypertensive patients, particularly if hypertensive encephalopathy exists (Table I). When the blood pressure is reduced, the cerebral vessels dilate, and cerebral blood flow and metabolism are maintained (Table II). When the blood pressure is reduced excessively, however, cerebral blood flow may decrease sharply. This is more frequently observed in the upright position when agents that block impulses at or beyond the ganglia are used, since reflexes responsible for adjusting peripheral vascular resistance to compensate for position change may be inhibited, with resultant excessive hypotension in the upright position. When this occurs, dizziness may be prominent. When, by excessive dosage, the supine blood pressure has been reduced to absolute normotensive levels with peripherally-acting agents, the patient should usually remain in bed; otherwise syncope may result when the patient stands up.

Generally, cardiac failure is improved following reduction of blood pressure. The initial response after administration of the ganglionic blocking agents, if heart failure is not present, is an acute reduction in blood pressure (Fig. 2) with a reduction in cardiac work load and little effect on cardiac output. However, due to release of venous tone, venous pressure also decreases, followed by a reduction in venous return and subsequently in cardiac output (Fig. 2).

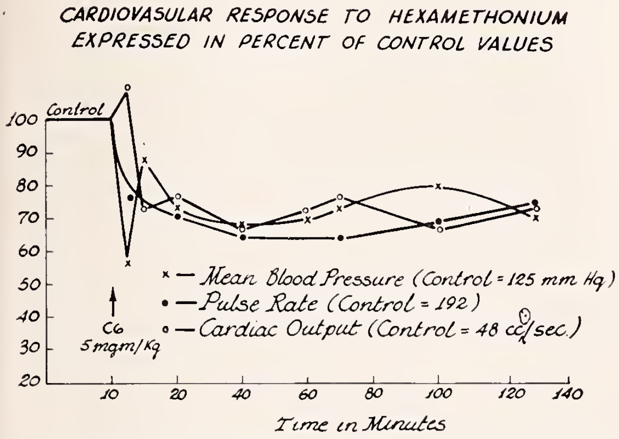


Fig. 2. Cardiovascular response to acute reduction in blood pressure with a ganglionic blocking agent, hexamethonium. There is at first a sharp decrease in arterial peripheral resistance. This is followed by venous dilatation, decreased venous return, and subsequently a reduction in cardiac output. (Courtesy: *Journal of Pharmacology and Experimental Therapeutics,* 106:157, 1952).

As a generalization, glomerular filtration rate and renal blood flow are depressed when the blood pressure is reduced acutely. One exception to this rule is hydralazine (Apresoline) which temporarily increases renal blood flow but does not alter or may reduce glomerular filtration rate (Fig. 3). Renal function is depressed (Fig. 4) more after the administration of the ganglionic blocking agents than it is following administration of the centrally-acting agents. When intravenous infusions of ganglionic blocking agents are continued, glomerular filtration rate and renal blood flow remain depressed throughout the period of infusion when the blood pressure is reduced to low normotensive levels. However, after intermittent parenteral administration (either intravenously or intramuscularly) there is a similar initial depression in renal function following which renal blood flow tends to return to control levels despite a persistent depression of glomerular filtration rate. After prolonged blood pressure regulation renal hemodynamic readjustment usually occurs following which renal function is no longer depressed (Fig. 4).

In patients with severe hypertension and associated renal disease, the reduction of the blood pressure must be approached cautiously. Patients with renal damage respond to blood pressure reduction in a way qualitatively similar to patients with normal kidneys, but the readjustment is

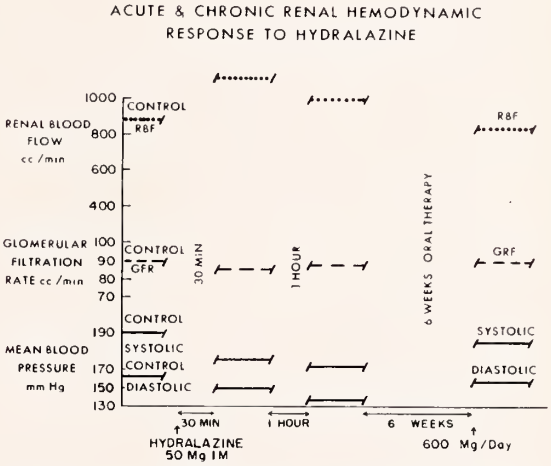


Fig. 3. Hydralazine administration increased renal blood flow at least temporarily. However, there was not a concomittant increase in glomerular filtration rate. Therefore, any beneficial effect on excretory function due to the Hydralazine on the kidney *per se* is not forthcoming.

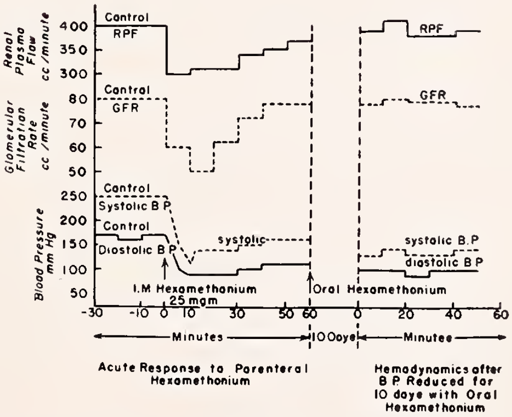


Fig. 4. The acute and chronic renal hemodynamic response to ganglionic blockade. There was a sharp reduction in glomerular filtration rate and renal blood flow. However, when the patient was later switched to oral administration and the drug was given over a long period of time, renal hemodynamic adjustment followed and despite the fact that blood pressure remained at normotensive levels, glomerular filtration rate and renal blood flow returned to the control levels. (Courtesy *American Journal of the Medical Sciences,* 225:379, 1953).

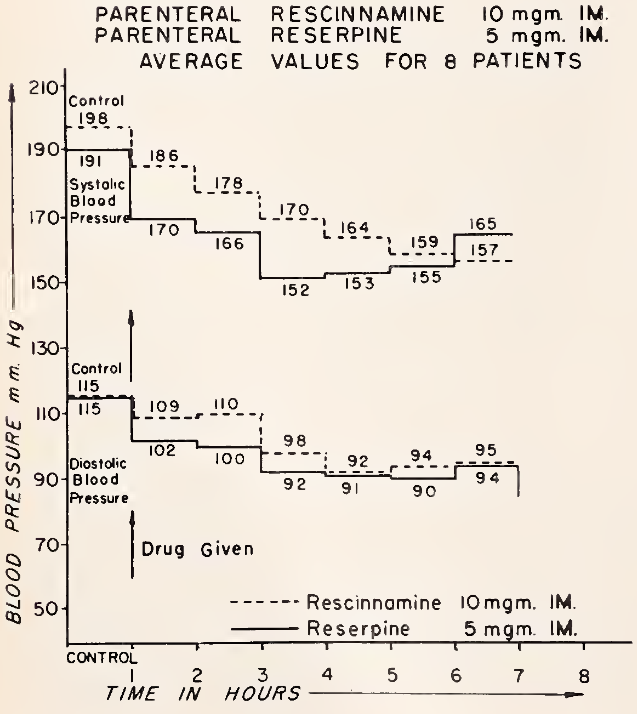
slower and incomplete. If readjustment is incomplete and if renal function is markedly impaired to begin with, even small reductions in glomerular filtration may be serious enough to produce renal decompensation. Incomplete renal hemodynamic readjustment is more likely to occur if blood pressure is suddenly reduced to absolute normotensive or hypotensive levels. This means that in patients with severe renal disease, extreme care must be taken in reducing the blood pressure. When the blood urea nitrogen (BUN) is 35 to 50 mg. prior to therapy, it is usually safe to acutely reduce the systolic pressure (to 160 to 180 mm. Hg.). If it is 50 to 80 mg. it is better not to reduce the systolic pressure much below 190 to 200 mm. Hg. When the BUN is above 80 to 100 mg. Hg., blood pres-

TABLE III. COMPARISON OF THE RENAL HEMODYNAMIC RESPONSE TO MECAMYLAMINE HEXAMETHONIUM AND PENTOLINIUM

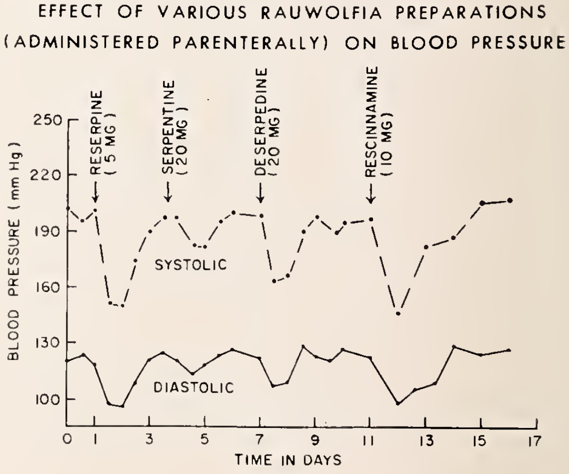
In Patients Receiving the Drugs for Prolonged Periods of Time

Drug	Number of Patients	Control	Drug	Per Cent of Control	P* Value	Tilt	Per Cent Drug Supine	P′ Value
Mean Blood Pressure* (mm.Hg.)								
Mecamylamine	8	154	135	88	.001	109	81	.001
Hexamethonium	7	159	134	84	.001	104	78	.001
Pentolinium	11	146	118	81	.001	—	—	—
Glomerular Filtration Rate (ml./min.)								
Mecamylamine	8	80	68	85	.2	50	74	.01
Hexamethonium	7	73	71	97	.1	56	79	.02
Pentolinium	11	83	80	96	.1	—	—	—
Renal Blood Flow (ml/min.)								
Mecamylamine	8	809	704	87	.3	508	72	.01
Hexamethonium	7	801	815	102	.1	501	61	.01
Pentolinium	11	866	963	111	.1	—	—	—

*Mean Blood Pressure=Diastolic pressure plus one-third of pulse pressure.
Control=Control recumbent.
Drug=Drug recumbent.
Per Cent Control=Drug/Control x 100.
Per Cent=Tilt/Drug x 100.
P* Value=Comparison of Control with Drug; A P Value of less than .05 is significant.
Tilt=Tilted head-up position for mecamylamine and ambulatory state for hexamethonium.
P′ Value=Comparison of Drug with Tilt.



5A



5B

Fig. 5A. A comparison of blood pressure response to Rescinnamine as compared to Reserpine. Although the dose requirement of Rescinnamine is one and one-half to two times that of Reserpine and the onset of action of Reserpine is less rapid, the degree of blood pressure reduction is equivalent. (Courtesy *American Journal of the Medical Sciences,* 231:542, 1956). B. Comparison of blood pressure response to four different alkaloid extracts of Rauwolfia given parenterally.

sure reduction can be rarely maintained without aggravating the renal failure. When encephalopathy is severe in these patients and if temporary reduction in blood pressure is mandatory, it is possible to lower the blood pressure intermittently but it is necessary to allow it to increase at regular intervals for the kidneys to function.

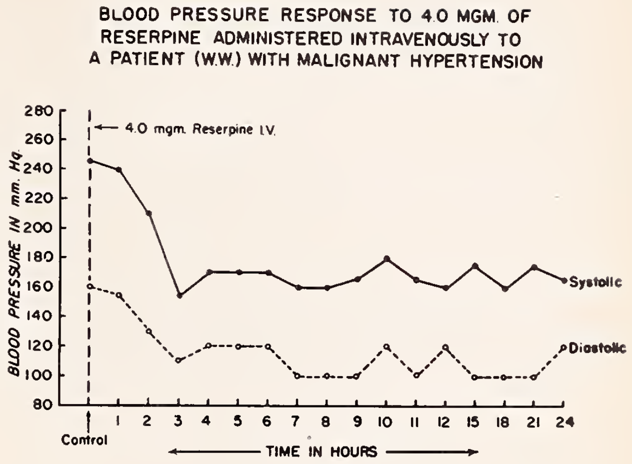
Clinical Use of Antihypertensive Drugs In Hypertensive Emergencies

Central Agents

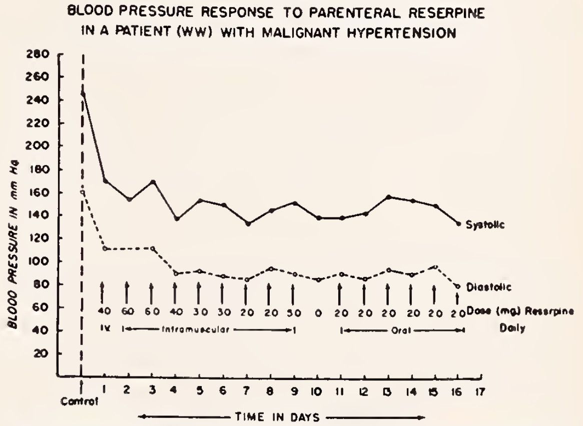
Rauwolfia Alkaloids.—Reserpine (Sandril, Serpasil, Serpiloid), and rescinnamine (Rescamine)—single alkaloids from Rauwolfia—when given in adequate doses parenterally, are probably the most useful antihypertensive agents available for the general treatment of hypertensive emergencies exclusive of the hypertension associated with pheochromocytoma. When given by the parenteral route, both agents are quite potent, in contrast to the response to the same drugs after oral administration (Figs. 5A and B). However, there is a latent period of one to two hours before the blood pressure decreases, following either intravenous or intramuscular administration.

Therefore, if immediate reduction in blood pressure is required, a more rapidly acting drug is indicated. Under these circumstances, in patients with a hypertensive emergency associated with essential hypertension, a ganglionic blocking agent or a Veratrum extract should be used initially and the reserpine or rescinnamine administered at the same time for maintaining the reduction in blood pressure. Following the initial reduction in blood pressure, these patients can usually be carried along with reserpine or rescinnamine, alone. Ganglionic blocking agents or Veratrum extracts are also indicated in patients who do not obtain an adequate reduction in blood pressure with parenteral reserpine.

In patients with hypertension association with acute glomerulonephritis and toxemia of pregnancy, reserpine in combination with hydralazine or Veratrum is the therapy of choice. We have studied reserpine administered parenterally to patients with hypertensive emergencies associated with severe essential hypertension, as well as in patients with severe hypertension of renal origin due to renal artery occlusion, cystic disease of the kidney, and chronic pyelonephritis. The results achieved indicate that reserpine, when administered parenterally, is a relatively potent antihypertensive agent and there are few hypertensive patients who do not obtain a significant reduction in blood pressure. The antihypertensive effect is manifested in the recumbent position as well as in the upright position, an attribute of considerable importance in the acutely ill and bedfast patient. The slow decrease in blood pressure which occurs and the relative rarity of excessive reduction allows effective and safe therapy without particularly close supervision by medical and nursing personnel.



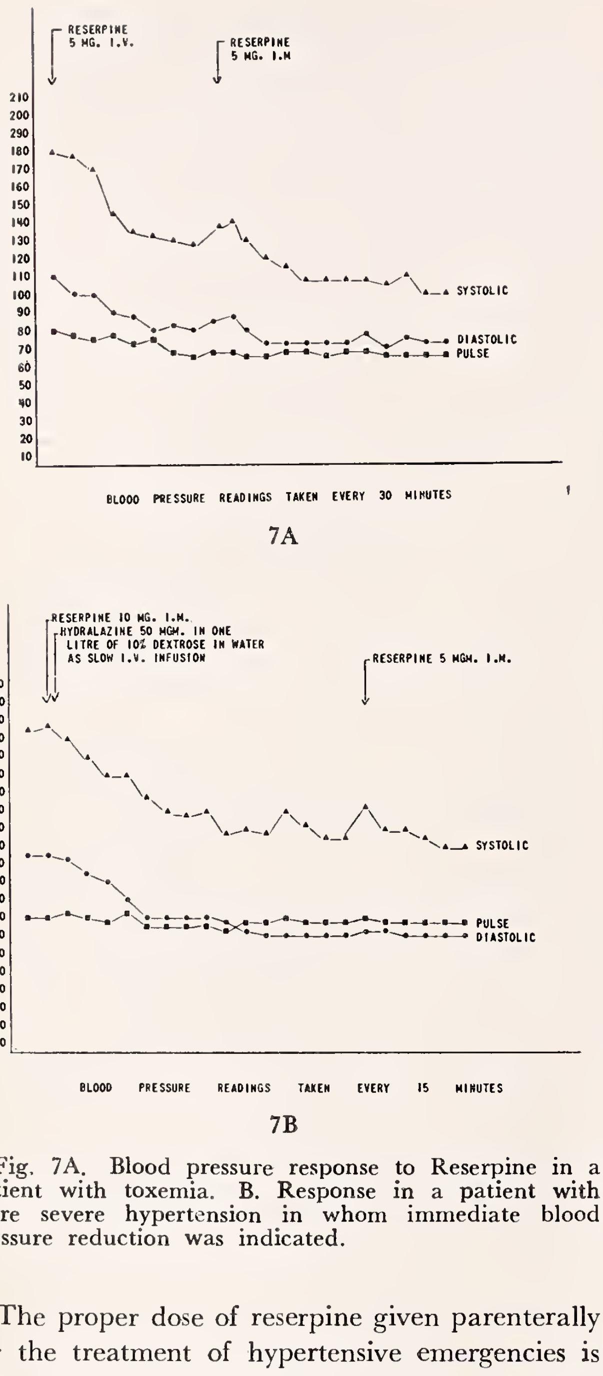
6A



(B) The blood pressure response during therapy with parenteral reserpine and after one week after conversion to the oral route. The blood pressure was maintained quite consistently at normotensive or mild hypertensive levels. One week following discontinuation of parenteral reserpine, pentolinium was added because of the gradual loss of adequate reduction in blood pressure.

6B

Fig. 6A. A patient with malignant hypertension. The blood pressure response to 4 mg. of Reserpine administered intravenously. The onset of significant blood pressure reduction occurred two hours after the injection, and maximum reduction occurred at three hours. Significant blood pressure effect was still present after twenty-four hours. B. The blood pressure response during therapy with parenteral Reserpine and for one week after conversion to the oral route. The blood pressure was maintained quite consistently at normotensive or mildly hypertensive levels. One week following discontinuation of parenteral Reserpine, Pentolinium was added because of a gradual loss of adequate reduction in blood pressure. (Courtesy *A.M.A. Archives of Internal Medicine,* 95:7, 1955).



7A

7B

Fig. 7A. Blood pressure response to Reserpine in a patient with toxemia. B. Response in a patient with more severe hypertension in whom immediate blood pressure reduction was indicated.

The proper dose of reserpine given parenterally for the treatment of hypertensive emergencies is best determined on the basis of the blood pressure response to increasing doses starting with 2.5 mg. and increasing the dose in 2.5 mg. increments until an adequate reduction in blood pressure is obtained (Fig. 6A). At least two hours must be allowed between doses in order to observe the maximum response to any single dose. The drug may be administered intramuscularly or it may be given intravenously in 100 cc. of 5 per cent glucose given over a fifteen to thirty-minute period. The response varies in degree and duration in different patients, but there is very little difference between the intravenous and the intramuscular route in the same patient.

After the proper dose has been established, the patients are then placed on a regular schedule which usually consists of 5 to 10 mg. of reserpine given every six to twelve hours (Fig. 6B). The maintenance dose and the interval between doses depends entirely on the blood pressure response and is adjusted so as to maintain the desired blood pressure level. When excessive reduction in blood pressure occurs, a vasopressor agent is an effective antidote. Blood pressure, pulse rate and respiration should be recorded every thirty minutes following the initial doses, and as necessary following subsequent doses.

The side effects tend to become more prominent as either the dose of reserpine or the frequency of administration is increased. Muscle tremors and other manifestations suggesting a Parkinson's syndrome may appear if doses of 5 to 10 mg. are given as frequently as every six hours for an extended period of time, but these disappear slowly after a reduction in the dose or frequency of administration, or after discontinuation of therapy. Doses in excess of 40 mg. a day should not be employed.

Rescinnamine is a pure alkaloid obtained from Rauwolfia serpentina, and is similar to reserpine. When given intramuscularly, rescinnamine is equally as effective as reserpine (Figs. 5A and B), but the dose required is one and one-half to two times as great. There is essentially no difference in the incidence and severity of side effects of reserpine as compared to rescinnamine except somnolence is less marked with the latter. The manner of administration is the same for both agents except that we employ an initial dose of rescinnamine of 5 mg. given intramuscularly. If this is inadequate, the dose is increased to a maximum of 15 mg. given no more frequently than every six hours. Doses in excess of 60 mg. a day should not be used.

Hydralazine (Apresoline).—In the treatment of hypertensive emergencies, this drug is limited primarily to use in acute glomerulonephritis and toxemias of pregnancy. Best results are obtained when it is used in conjunction with reserpine. The onset of reduction in blood pressure (fifteen to twenty minutes) is more rapid after hydralazine than after the administration of reserpine. Therefore, it is given in cases with acute nephritis and toxemia of pregnancy where rapid onset of action is necessary, or in patients who do not respond adequately to reserpine alone.

TABLE IV. GENERAL OUTLINE OF ANTIHYPERTENSIVE THERAPY FOR THE TOXEMIA PATIENT CONFINED TO THE HOSPITAL

Severity of Disease	Hypotensive Drug Therapy	
	Reserpine	Hydralazine
Mild pre-eclampsia (blood pressure 140/90 to 160/100)	5 mg. I.M. or I.V.—may be repeated in 1-2 hours if still hypertensive. Thereafter, for maintenance, 5 mg. every 4-6 hours as needed for blood pressure of 140/90 or above.	Given only when normotensive response not obtained with reserpine alone—may give 5-10 mg. I.V. as single injection or slow I.V. infusion with 25 mg. placed in one liter of 10% dextrose in water.
Severe pre-eclampsia (blood pressure above 160/100)	10 mg. I.M. or I.V.—may be repeated in 1-2 hours if still hypertensive. Thereafter, for maintenance 5 mg. every 4-6 hours as needed for blood pressure of 140/90 or above.	10 to 20 mg. I.V. as single injection for rapid initial blood pressure fall if desired response not obtained from reserpine—then repeat only when unable to maintain normotensive on reserpine alone. May be given by continuous I.V. infusion, 50 mg. placed in one liter of 10% dextrose in water, when blood pressure labile or is difficult to get desired response as in labor.
Severe pre-eclampsia and primary hypertensive disease (blood pressure 160/100)	Same as for severe pre-eclampsia.	Same as for severe pre-eclampsia.
Eclampsia	10 mg. I.V. and repeat in 1-2 hours if still hypertensive; then give maintenance dose of 5-10 mg. every 4-6 hours to ensure sedation and a normotensive state.	If BP 170/100 or above, give 20 mg. single I.V. injection. If BP between 150/90 and 170/100, give 10 mg. single I.V. injection. If BP between 130/80 and 150/90, use continuous I.V. infusion of 25 mg. in one liter of 10% dextrose in water slowly, so that it may be discontinued quickly should the BP fall too low. If after the initial injections, the hypertension is very labile or difficult to control the continuous I.V. infusion of 50 mg. in one liter of 10% dextrose in water should be used.

TABLE V. RESPONSE OBTAINED WITH RESERPINE ALONE AND IN COMBINATION WITH HYDRALAZINE
303 Patients with Toxemia of Pregnancy

Diagnosis	Total Number Patients	Good		Fair		Poor	
		Number	Per Cent	Number	Per Cent	Number	Per Cent
Severe pre-eclampsia	95	85	89	10	11	00	0
Primary hypertensive disease with severe pre-eclampsia	25	23	92	2	8	0	0
Mild pre-eclampsia	169	153	91	16	9	0	0
Eclampsia	14	14	100	0	0	0	0
Total	303	275	91	28	9	0	0

When hydralazine is administered in patients with hypertension due to toxemia of pregnancy, an initial dose of 5 to 20 mg. should be given intravenously as a single injection over a five-minute period or intramuscularly in a dose of 10 to 25 mg. Then 25 to 50 mg. (depending on severity) should be placed in 1000 cc. of 10 per cent glucose in water and be given by continuous intravenous infusion (Table IV). The rate of infusion should be adjusted depending on the blood pressure response. When given parenterally, there is usually a pronounced fall in bood pressure within twenty minutes, the reduction being most marked in the diastolic pressure. The effect may last up to twelve hours. The general approach to antihypertensive therapy in toxemia of pregnancy is outlined in Table IV.

Of 303 patients with toxemia of pregnancy treated by the author and his colleagues, 211 responded adequately to parenteral reserpine, alone (Table V). Eighty-nine patients received a combination of reserpine and hydralazine. With this approach, blood pressure has been well controlled in over 90 per cent of patients (Table V).

TABLE VI. PARENTERAL RESERPINE
Approximate Individual Dose

Average Age Years	Weight		Dose—Mg.	
	Pounds	Kilograms	Average	Maximum*
1	22	10	1.0	1.5
3	34	15	1.5	2.0
5	44	20	2.0	3.0
9	66	30	3.0	4.5
12	88	40	4.0	6.0
14	110	50	5.0	7.5
Adult	150	70	7.0	10.0

*This is the maximum effective dose.

In patients with acute nephritis, the blood pressure may be elevated initially or it may not appear until the third or fourth day of the disease. In our experience, the ideal drug for reducing the blood pressure in patients with acute nephritis is parenteral reserpine used alone or in combination with hydralazine. The reserpine is given intramuscularly as described previously. In children the dose is usually regulated according to the weight of the child (Table VI). The blood pressure is usually reduced to a safe range, but rarely does it become entirely normal. When adequate antihypertensive effect is not obtained with reserpine alone, then a supplement of hydralazine should be added, but the reserpine continued as the basic agent.

In certain patients parenteral reserpine either fails to give ideal antihypertensive effect (Fig. 8) or else the critical state of the patient's cardiovascular reserve demands a more rapid reduction of blood pressure. The addition of single doses of hydralazine is usually effective. This combined approach permits the use of smaller, and therefore safer, doses of hydralazine.

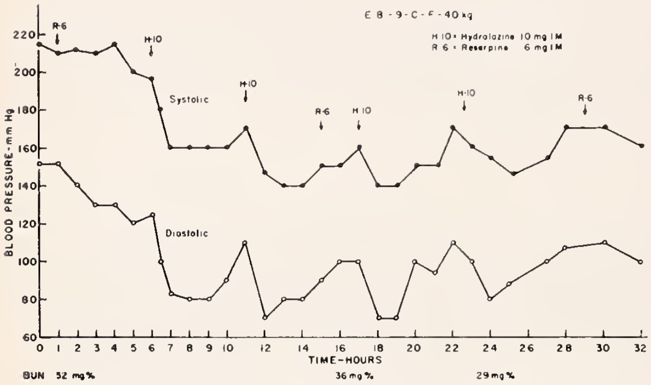


Fig. 8. Patient with acute nephritis. The response to Reserpine (R-6) was not adequate and it became necessary to administer Hydralazine (H-10).

In the patients who have already received reserpine, and who show inadequate antihypertensive effect, it is our custom to begin by administering a 5 mg. dose of hydralazine (intravenously or intramuscularly). Then the blood pressure is measured every fifteen minutes. If less than the desired fall in bood pressure occurs within forty-five minutes, the supplemental dose of hydralazine is increased by 5 mg. increments until blood pressure control is achieved (Fig. 9). The dose thus established is used thereafter as often as necessary, as a supplementary drug to control blood pressure. In this way, a titration of the effective dose is carried out *in vivo*. We have preferred the intramusclular administration of hydralazine. In the patient who is admitted with convulsions, or other evidence of hypertensive encephalopathy, the simultaneous administration of intravenous reserpine and hydralazine may be necessary.

Veratrum Drugs.—When a ganglionic blocking agent (when used in combination with parenteral reserpine) is also ineffective in hypertensive emergencies one can then use a continuous intravenous infusion of alkavervir (veriloid) or protoveratrine (veralba). Such Veratrum extracts are the most potent antihypertensive agents available today, and it is rare that a patient will not respond to these drugs. However, because they usually produce nausea and vomiting and because of their potency and the difficulty of regulating the blood pressure when these agents are used, they should be employed only as a last resort and only in those patients who do not respond to more simple methods of treatment. These drugs are as effective in the supine as in the upright position, a virtue of note.

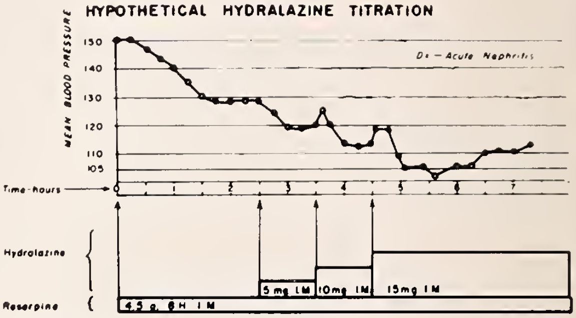


Fig. 9. Dose titration of Hydralazine in children with acute nephritis.

Parenteral alkavervir (veriloid) is one of the most dependable centrally-acting veratrum extracts which can be given either intramuscularly or by continuous intravenous infusion. The onset of action is rapid, that is, within several minutes. When using this drug, great caution must be exercised because the blood pressure can easily be lowered excessively. It also produces a rather marked reduction in pulse rate by vagal stimulation (Fig. 10). It may produce nausea and vomiting in many cases in which an effective reduction of blood pressure has been obtained. The incidence of side effects is high when adequate amounts of this agent are used to reduce the blood pressure to normotensive levels (Table I). For example, in one study conducted by the author, nausea or vomiting was observed in seven out of seventeen patients. Despite the high incidence of side reactions, some improvement in the encephalopathic manifestations exhibited by these patients was observed in all but four patients and in seven of the seventeen cases, all manifestations of encephalopathy cleared following an effective and maintained reduction in blood pressure (Table I).

When used intravenously, an initial priming dose of alkavervir is given which consists of 0.5 microgram per kilogram body weight per minute over a twenty-minute period (Fig. 10). The blood pressure should be checked every minute during this time and until an adequate reduction in blood pressure is obtained. A change is then made to the sustaining infusion (Fig. 11). The priming dose is prepared by diluting 10 micrograms per kilogram body weight of intravenous solution of alkavervir in 20 cc. of 5 per cent glucose in distilled water. If the desired hypotensive effect has not been achieved with the initial priming dose, it may be repeated at least in part. The sustaining solution consists of 4 mg. of alkavervir added to a liter of 5 per cent glucose in distilled water. This is given at a rate that is regulated depending on the amount necessary to maintain the desired blood pressure level. As a precaution, a similar infusion containing 4 mg. of norepinephrine per liter of 5 per cent glucose in distilled water should be prepared beforehand and connected through a Y-Tube (Fig. 11) for immediate infusion should the blood pressure decrease excessively at any time. Norepinephrine is a very effective antidote.

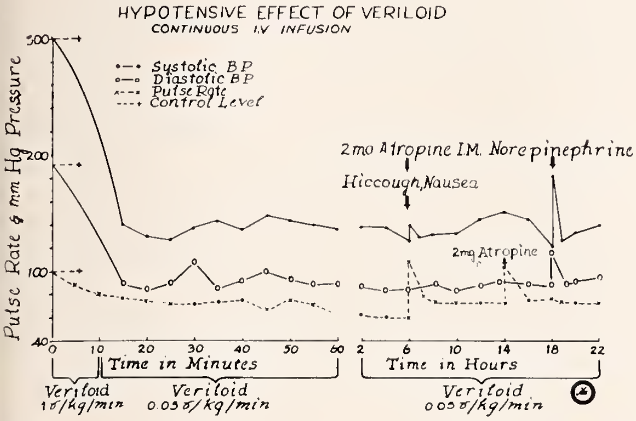


Fig. 10. Blood pressure response to alkavervir (Veriloid) administered intravenously in a patient with malignant hypertension.

During the initial period of adjusting the rate for a sustained infusion of alkavervir or protoveratrine, the blood pressure and pulse rate should be checked every two or three minutes for the first thirty minutes and then every five minutes until the blood pressure is completely stabilized. The blood pressure should be checked every fifteen to twenty minutes throughout the entire period of intravenous infusion. This requires the full-time attendance of a nurse or someone well trained in the use of these drugs who is capable of taking blood pressures properly. As soon as feasible, oral or intramuscular hypotensive therapy should replace the intravenous infusion (Figs. 12A and B).

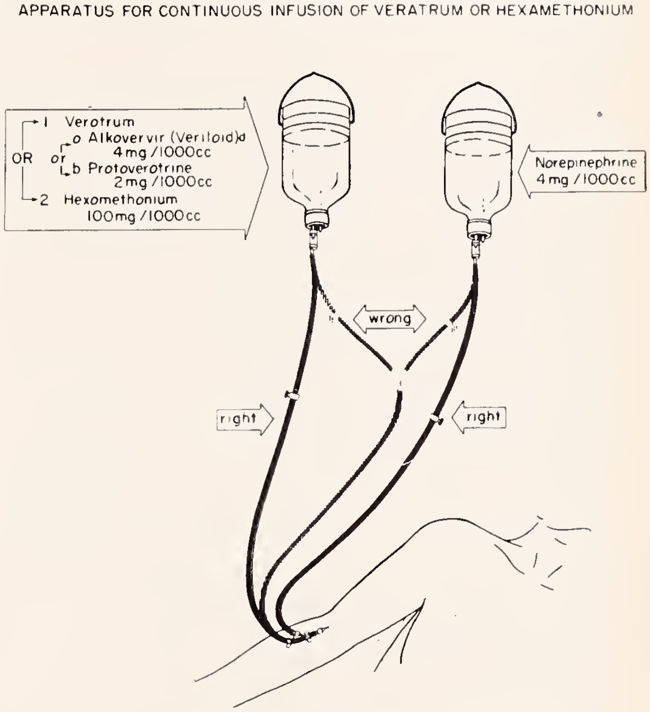
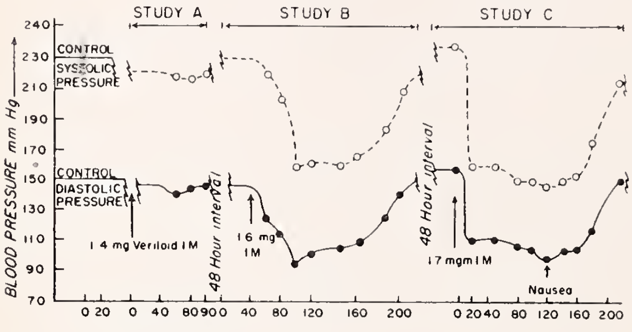


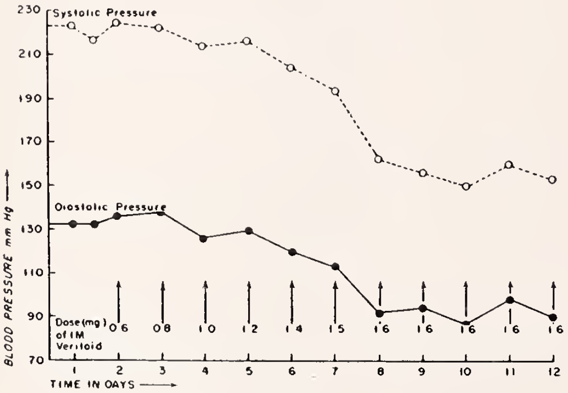
Fig. 11. Method of setting up a continuous infusion for the administration of Veratrum or ganglionic blocking agents. When using either alkavervir or protoveratrine, it is necessary to have a vasopressor agent available for immediate administration should excessive reduction in blood pressure occur.

When the route of administration of alkavervir is changed from the intravenous to the intramuscular one, the first intramuscular dose should be 0.6 mg. given every four to six hours (Figs. 12A and B). The dose should then be increased in 0.2 mg. increments until significant reduction in blood pressure occurs (Figs. 12A and B) after which the dose should be increased more gradually (usually in about 0.1 mg. increments) until the blood pressure is reduced to the desired level. Then the frequency of administration is adjusted depending upon the length of action of the drug as reflected in the length of time the pressure remains reduced. The blood pressure should be recorded at least every fifteen minutes until the dose is well adjusted following intramuscular administration of the drug. Norepinephrine or a similar vasopressor agent should be available at the bedside at all times in the event that excessive hypotension occurs. Atropine, in a dose of 1 mg. is very effective in combatting excessive bradycardia. There is no effective antidote for the nausea and vomiting that may be associated with veratrum administration.



12A

BLOOD PRESSURE RESPONSE TO A TITRATED DOSE OF INTRAMUSCULAR VERILOID (AQUEOUS SOLUTION) WITHOUT PREVIOUS ORAL THERAPY



12B

Fig. 12A. When the blood pressure is brought under control with an intravenous infusion of Veratrum, then the drug can be administered by the intermittent intramuscular route. When this is done the dosage must be titrated until an effective reduction in blood pressure has occurred. B. Demonstration of dosage titration of Veriloid in overall blood pressure response of a patient with malignant hypertension. (Courtesy *American Journal of the Medical Sciences,* 226:477, 1953).

Intravenous protoveratrine (veralba 0.2 mg./cc.) is probably as effective as alkavervir. The priming solution is made up by diluting 0.5 cc. (0.1 mg.) of protoveratrine in 10 cc. of 5 per cent glucose. This diluted solution is then injected at a rate of 0.5 cc./minute. If there is no fall in blood pressure after five minutes, the procedure is repeated. When the desired decrease in blood pressure has been obtained, a continuous intravenous infusion of 2 mg. of protoveratrine (one ampule) in 500 cc. of glucose (5 per cent) solution is then administered. The infusion rate is adjusted as necessary in order to achieve the desired blood pressure level without vomiting—usually 40 to 80 drops per ten minutes. The blood pressure should be checked at least every fifteen minutes after the blood pressure has become stabilized.

After the immediate emergency is over, the intramuscular route of administration may be employed. For this purpose, give 0.6 cc. (0.12 mg.) intramuscularly and check the blood pressure every fifteen minutes. The maximum effect occurs in one to two hours. Repeat with increments of 0.2 cc., added or reduced, every four to six hours depending on the patient's response. Atropine is effective for the bradycardia but not for nausea or vomiting. If excessive reduction in blood pressure occurs, a vasopressor drug is effective.

Ganglionic Blocking Agents

When reserpine (administered parenterally) is ineffective in patients with other than acute nephritis and toxemia of pregnancy, then a ganglionic blocking agent such as hexamethonium, pentolinium (Ansolysen), trimethaphan camphorsulfonate (Arfonad) or mecamylamine (Inversine) should be tried either alone or in combination with reserpine. When given intramuscularly, they are safe and usually effective therapeutic agents, and the danger of excessive reduction in blood pressure is not as great as with Veratrum administered parenterally. The frequency of administering intramuscular ganglionic blocking agents will depend on the length of time that the blood pressure remains reduced following each dose. As soon as the hypertensive emergency is under control, the patient should be started on oral therapy.

As in ambulatory patients, care must be exercised in reducing the blood pressure in patients with renal failure lest the renal function be further impaired. The best estimate of adequate renal functional capacity is gained by repeated determinations of the blood urea nitrogen. If the blood pressure is reduced excessively, it can be corrected by the administration of a vasopressor agent such as norepinephrine (Fig. 13) or any other readily available vasopressor agent that can be given intravenously or intramuscularly.

The blood pressure response to ganglionic blocking drugs is most marked in the standing position, which is one of their greatest shortcomings.

Thus, these agents are of limited value in the bedfast patient who is supine. In patients with severe disease, an adequate reduction in blood pressure is sometimes impossible, necessitating the use of Veratrum extracts. It is not good to give excessive doses of ganglionic blocking agents with the idea of "forcing the blood pressure down" since frequently the result will be a severe ileus due to blockade of the parasympathetic ganglia without obtaining the desired reduction in blood pressure.

Assistance in blood pressure reduction can be obtained if the head end of the bed is raised on blocks. Usually 10 to 12 inch blocks are effective, but the farther the head end of the bed is raised, the better. If large doses of the drug have been given and a marked orthostatic effect obtained, careful attention must be given to the degree of blood pressure reduction obtained when the patient is tilted. Otherwise, excessive hypotension can occur, resulting in cerebral ischemia and cerebral anoxia.

When constipation becomes a problem, cathartics and cholinergic agents should be used freely. Cascara sagrada in a dose of 15 cc. to 30 cc. or milk of magnesia in doses of 30 to 60 cc. is usually adequate. Cholinergic agents are sometimes more effective than cathartics. For this purpose, 15 to 45 mg. of prostigmine or 5 to 10 mg. of pilocarpine given orally three or four times a day is usually adequate. If the patient is comatose, 1 mg. of prostigmine is used subcutaneously; when ileus is present, 1 mg. should be given every hour until peristalis returns. When treating hypertensive emergencies, mecamylamine is of limited value for bedfast patients because of the persistence of the blockade and resultant constant and frequently severe constipation. When this drug is used, particular attention must be given to the bowels, and cathartics or cholinergic agents must be used freely.

When giving hexamethonium by continuous intravenous infusion for hypertensive emergencies 100 to 150 mg. are placed in 1000 cc. of 5 per cent glucose in distilled water. The drug is infused at a moderate rate until the blood pressure begins to fall, then the rate of infusion is adjusted so as to maintain an adequate reduction in blood pressure. As a safety precaution the infusion apparatus should be connected to a Y-tube such as in Fig. 11. Norepinephrine is an effective antidote for hexamethonium, as well as all other ganglionic blocking agents, should excessive reduction in blood pressure occur. The blood pressure and pulse rate should be checked at least every five minutes during the initial period of adjustment. After the blood pressure has been stabilized it should be checked every fifteen to twenty minutes.

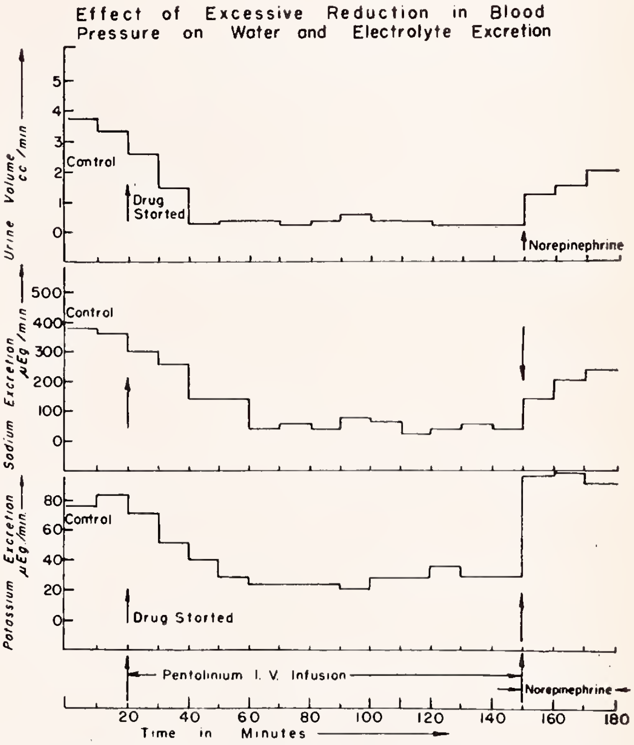


Fig. 13. A patient in whom an excessive reduction in blood pressure was observed with Pentolinium (Ansolysen). The patient became anuric due to a marked decrease in glomerular filtration rate. However, as the blood pressure was elevated with a vasopressor agent, glomerular filtration rate returned toward control levels and urine output also returned to previous values.

When administering hexamethonium intramuscularly, the initial dose is 15 milligrams. After waiting one hour to determine the effect of the initial dose, another dose is given, and subsequent doses are given in increasing amounts until an adequate effect is obtained. Usually, the drug can be repeated every one to two hours until effective reduction in blood pressure occurs. Doses in excess of 100 mg. are no more effective than this amount. The frequency of administering intramuscular hexamethonium will depend on the length of time that the blood pressure remains reduced after each injection. Usually, it is only necessary to give the drug every four to six hours. As soon as the hypertensive emergency is under control, the patient should be started on oral therapy. Care must be exercised in reducing the blood pressure for patients with renal failure lest the renal function be impaired further. A systolic blood pressure of 170 to 180 mm. Hg. is usually a safe level unless the BUN is markedly elevated prior to therapy.

TABLE VII. GANGLIONIC BLOCKING AGENTS

Summary of Results in Patients Treated for Fulminating Heart Failure Associated with Severe Hypertension

	Hexamethonium	Mecamylamine (Inversine)	Arfonad
Number patients treated	16	8	11
Significant reduction in blood pressure	12	6	10
Normotensive	4	2	9
Improvement in pulmonary edema	13	7	10
Recovery	12	6	10

Pentolinium is used in essentially the same manner as hexamethonium. When making up the intravenous infusion, 50 to 75 mg. per 1000 cc. of 5 per cent glucose is adequate. The rate of infusion is adjusted according to the blood pressure response. When giving the drug intramuscularly, an initial dose of 5 mg. is used. After one to two hours, the second dose is given, and so on until an adequate reduction in blood pressure is obtained. Usually, the second dose is 10 mg. and the subsequent doses are progressively increased until the blood pressure has been adequately controlled or a maximum of 50 mg. per dose is given. If the blood pressure is not reduced adequately with 50 mg. per dose or less, given intramuscularly, it is rarely possible to obtain an increased response with doses in excess of this amount. The same problems exist with this drug as have been noted in the use of hexamethonium.

Mecamylamine is completely absorbed from the gastrointestinal tract and consequently is as effective when given orally as when given parenterally. Therefore, when the patient is capable of taking medication orally, he can be given the same dose by this route as was found to be effective previously when given by the parenteral route. Complete absorption also allows reproducible blood pressure responses to equal doses of drug with minimum day-to-day variation, the lack of which is a serious drawback to the use of ganglionic blocking agents of the quaternary ammonium variety, such as hexamethonium and pentolinium.

The initial dose of mecamylamine is 3 mg. given intramuscularly or subcutaneously. This can be repeated every two hours and increased in 3 to 5 mg. increments until an adequate reduction in blood pressure is obtained. Doses in excess of 50 mg. produce no additional effect and should not be given. As with the other blocking agents, tilting the bed in the head-up position assists in reducing the blood pressure. The frequency of administration will depend on the length of time the drug remains effective, frequently twelve to eighteen hours. As with pentolinium and hexamethonium, if the blood pressure is reduced excessively, the glomerular filtration rate may become markedly depressed. Therefore, when this occurs, it is necessary to administer a vasopressor agent.

Ganglionic blocking agents are particularly useful in patients with severe and acute heart failure associated with sudden elevation in blood pressure (Table VII). As therapeutic blockade is established, there is a reduction in venous tone, resulting in a decrease in venous pressure and venous return, followed by an increase in cardiac output. The relief of pulmonary edema is often dramatic.

In hypertensive patients without heart failure, cardiac output decreases, apparently due also to a decrease in venous tone and subsequent reduction in venous return. The significance of the reduction in cardiac output and untoward effects resulting therefrom in the patient without heart failure are not known to me. Certainly there is no harm done since I am not aware of aggravation or precipitation of heart failure from the judicious use of ganglionic blocking agents.

When used in patients with hypertension and fulminating heart failure, hexamethonium or pentolinium can be given intravenously in a concentrated solution in order to avoid extra fluid administration (Figs. 14A and B). An initial dose of 25 mg. of hexamethonium or 10 mg. of pentolinium in 1 to 10 cc. of fluid is usually preferable. This dose can be increased and repeated after thirty minutes or as frequently as necessary to reduce the blood pressure. After an effective blood pressure reduction has been obtained, hexamethonium must usually be given every two to four hours, pentolinium every six to eight hours, and mecamylamine (5 to 10 mg.) every eight to ten hours. For maintenance therapy, the drugs are usually given intramuscularly.

Arfonad (trimethaphan camphorsulfonate) is useful in patients with fulminating pulmonary edema because it is more effective in reducing the arterial blood pressure than the other ganglionic blocking agents (Table VII). An additional attribute is that it is effective for only short periods of time so that the degree of blockade and blood pressure reduction can be regulated from minute to minute. However, it must be given as a continuous intravenous infusion.

Peripherally Acting Agents

Phenoxybenzamine is an effective adrenergic blocking agent which can be given intravenously (but not intramuscularly). However, because of the complete blockade (both humoral and at the end organ) produced by this drug, rendering vasopressor agents ineffective, it is generally thought inadvisable to administer this blocking agent by the parenteral route. Should excessive reduction in blood pressure occur with the use of this agent, no effective antidote is available. When its use becomes mandatory, 30 mg. is placed in 500 cc. of 5 per cent glucose in distilled water. Should excessive hypotension occur, the patient should be tilted in a 20 to 25 degree head-down position and the drug should be discontinued. *Do not infuse norepinephrine since this drug will only potentiate the hypotension.*

When taken by mouth, phenoxybenzamine is absorbed rapidly, and the effect lasts twelve to eighteen hours. It is useful in a few cases of severe hypertension and in the temporary management of patients who are not yet ready for surgery but who suffer from severe hypertension due to pheochromocytoma.

Phentolamine (Regitine) is useful for the treatment of patients with severe hypertension due to the liberation of norepinephrine by a pheochromocytoma. When used for this purpose, it is administered as a temporary measure for controlling the blood pressure prior to and during surgical removal of the tumor. A dose of 5 mg. of phentolamine is given intravenously or intramuscularly and is repeated as frequently as necessary in order to control the blood pressure.

If for any reason the patient must be treated for prolonged periods, the blood pressure can usually be better controlled with phenoxybenzamine given by the oral route. The dose of phenoxybenzamine by this route is 10 mg. every six hours. The dose is then subsequently increased in 10 mg. increments until effective reduction of the blood pressure is obtained. This method of therapy should be considered a temporary expedient only, since this is a curable disease, and the tumor should be surgically removed as soon as possible.

Ganglionic blocking and centrally-acting antihypertensive agents are not only ineffective in treating pheochromocytoma, but may actually precipitate an acute rise in blood pressure since these agents do not block the ability of norepinephrine to constrict the arterioles.

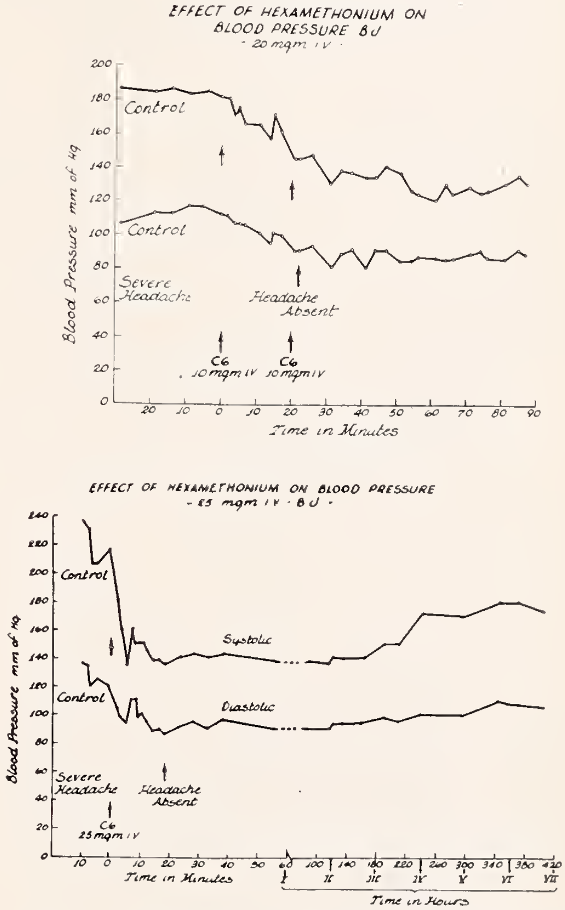
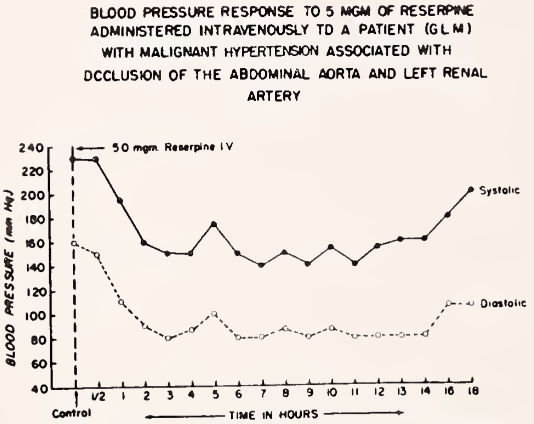


Fig. 14A. Blood pressure response to hexamethonium in a patient with mild hypertension. B. Blood pressure response to hexamethonium in a patient with severe hypertension.

Special Considerations

Primary Renal Disease (Excluding Nephritis).—As indicated previously, even the patient with primary renal disease may benefit from blood pressure control if severe hypertension becomes an important component of his disease. A case example is summarized in Figure 15. This was a patient who developed marked atherosclerosis of the aorta which also involved the right renal artery resulting in acute occlusion of the artery due to thrombus formation. The left renal artery was not involved.

At the time of admission to the hospital, the patient had marked encephalopathy, retinal hemorrhages, papilledema, and he was convulsing as a result of malignant hypertension, probably secondary to the ischemic kidney ("Goldblatt Kidney"). He was placed on parenteral reserpine given every six hours. This effectively reduced the blood pressure, and most of the cerebral manifestations subsided. He was gradually switched to a combination of oral Rauwolfia and a ganglionic blocking agent, mecamylamine. The aorta was replaced by a homograft but the kidney was not removed due to the poor condition of the patient. The postoperative course was uneventful and the blood pressure has since been well regulated for a period of three years and the patient is now in full-time employment.



RENAL FUNCTION RESPONSE FOLLOWING HOMOGRAPH IN PATIENT WITH GOLDBLATT KIDNEY

	Glomerular Filtration Rate ml/min.		Renal Blood Flow ml/min.	
	Right (occluded)	Left	Right (occluded)	Left
Control	0	25	0	130
Blood Pressure Rx.	0	35	0	200
Homograph				
1 month	0	45	0	425
6 month	0	60	0	725

Fig. 15. Blood pressure response in a patient who had a sudden onset of malignant hypertension due to occlusion of one renal artery. This patient responded to Reserpine in the same way that a patient with essential hypertension does. As the blood pressure remained reduced, glomerular filtration rate in the unoccluded kidney increased and after six months of therapy the glomerular filtration rate and renal blood flow had returned to normal for one kidney. The blood urea nitrogen over this period of time decreased from a level of 70 mg. per cent to 30 mg. per cent. (Courtesy *A.M.A. Archives of Internal Medicine,* 98:427, 1956).

This patient had no return of renal function in the occluded kidney (right). However, in the opposite kidney (left), glomerular filtration rate and renal blood flow, which were depressed to less than 50 per cent of normal for one kidney prior to therapy, gradually increased so that now they are approximately normal. His blood urea nitrogen has decreased from 70 to 30 milligrams per 100 cc. of blood.

Undoubtedly the depression in function in the unoccluded left kidney was due to severe vasoconstriction, intrarenal hemorrhages, and acute vascular damage, rather than chronic deterioration. Since the process was relatively acute, it was partially reversible. Had the hypertension and increased sympathetic vasoconstrictor response been allowed to continue, progressive renal failure would have developed had the patient not died first of cerebral manifestations.

Therefore, even in the patient with primary renal disease and hypertension secondary thereto, benefit can be derived from effective antihypertensive therapy. These patients should be treated in the same way as patients with hypertension associated with so-called essential hypertension are treated. If these patients are first seen in a state of crisis, they should receive parenteral reserpine. If more potent agents are necessary, one of the parenterally-administered Veratrum Compounds is used.

As soon as the emergency is brought under control, the diseased kidney should be removed if the problem is that of unilateral disease. If surgery is not feasible (as in the case of cystic disease of kidney or chronic pyelonephritis), the patient should be placed on a regimen of Rauwolfia given orally in combination with a ganglionic blocking agent and treated similarly to the patient with severe essential hypertension. Naturally, in the case of chronic pyelonephritis, it is mandatory that the etiologic organism be identified and treated with a specific antimicrobial agent.

Pheochromocytoma.—This has been described adequately elsewhere in this symposium.

Acute Steroid Hypertension.—The best treatment of acute hypertension associated with steroid administration is withdrawal of the steroid. However, in patients with severe and malignant disease such as lupus erythematous and leukemia, this is not possible. Therefore, it sometimes becomes necessary to continue the steroids and treat the

TABLE VIII. OUTLINE FOR ANTIHYPERTENSIVE TREATMENT OF HYPERTENSIVE EMERGENCIES

Emergency	Initial Therapy				Adjunctive Therapy When Initial Drug Inadequate	Maintenance Therapy After Emergency Over
	Drug	Dose	Route	Frequency		
Encephalopathy	Reserpine or Rescinnamine	2.5 to 10 mg. 5 to 15 mg.	I.M. I.M.	4-12 hours 4-12 hours	Hexamethonium 10-100 mg. I.M. If inadequate then veriloid titration I.V. infusion	Rauwolfia+ganglionic blocking agent
Fulminating heart failure	Hexamethonium or Arfonad	10 to 100 mg. Titration	I.M. I.V.	30 min. to 4 hours Continuous infusion	Reserpine I.M. or veriloid titration by I.V. infusion	Rauwolfia+ganglionic blocking agent
Intractable angina with severe hypertension	Hexamethonium		I.M.	1-4 hours	Reserpine 2.5 to 10 mg. given I.M.	Rauwolfia+ganglionic blocking agent
Hypertensive crisis	Reserpine or Rescinnamine	2.5 to 10 mg. 5 to 15 mg.	I.M. I.M.	4-12 hours 4-12 hours	Hexamethonium 10-100 mg. I.M. If inadequate response then veriloid by I.V. infusion	Rauwolfia+ganglionic blocking agent
Malignant hypertension of renal origin*	Reserpine or Rescinnamine	2.5 to 10 mg. 5 to 15 mg.	I.M. I.M.	4-12 hours 4-12 hours	Hexamethonium 10-100 mg. I.M. If inadequate response then veriloid by I.V. infusion	Rauwolfia+ganglionic blocking agent
Severe steroid hypertension	Reserpine	2.5 to 10 mg.	I.M.	4-12 hours	Veratrum, inversine, dibenzyline	Reserpine+veratrum+inversine and dibenzyline as required
Pheochromocytoma	Regitine	5 mg.	I.M.	p.r.n.	Dibenzyline 10-20 mg. orally	Surgery
Renal failure (uremia)	None				None	None

*Cystic disease, pyelonephritis, renal artery occlusions, etc.

hypertension as it develops. None of the current programs are very effective.

Reserpine should be given a trial. As in the treatment of hypertensive emergencies associated with essential hypertension, if this is not adequate, parenteral Veratrum should be used. Ganglionic blocking agents appear to be the least useful for this therapeutic problem.

As soon as the blood pressure is brought under control, oral therapy should be instituted. Alseroxylon (Rauwiloid) in combination with Veratrum or a ganglionic blocking agent is most useful. The Veratrum should first be given a trial, and the dose must be titrated. If nausea and vomiting ensue before the blood pressure effect is obtained, it is then necessary to use a ganglionic blocking agent. The dose is also established by titration. A combination of centrally-acting agents, with a ganglionic blocking agent, and an adrenergic blocking agent will sometimes be effective when the usual methods have failed. Each of the three agents is titrated up to tolerance or until the blood pressure is reduced adequately. Recently, we have observed that chlorothiazide (Diuril) given in a dose of 500 mg. every twelve hours will markedly increase the effectiveness of these agents.

Summary

In the approach to a patient with a hypertensive emergency associated with severe essential hypertension, it is necessary first to appraise the state of renal compensation, particularly if cerebration is abnormal. This can best be done with an estimate of the blood urea nitrogen (BUN). If the blood urea nitrogen is normal, renal failure is not likely to be responsible for any disturbances in the sensorium. Retinal examination is essential also. Retinal hemorrhages indicate the degree of general arteriolar damage. Papilledema suggests increased intracranial pressure and cerebral edema, which frequently accounts for derangement of cerebral function in patients who do not have associated renal failure.

After the state of renal function is known, therapy should be approached systemically (Table VIII). If immediate blood pressure reduction is not indicated and a delay of two to three hours is possible, parenteral reserpine or rescinnamine is the drug of choice. The initial dose is 2.5 mg., repeated in two hours if blood pressure reduction is not adequate. When the blood pressure is not reduced adequately at a maximum dose of 10 mg. of reserpine or 15 mg. of rescinnamine, given every six hours, a ganglionic blocking agent should be tried. There is very little difference in responsiveness among the different ganglionic blocking agents. I prefer hexamethonium for initial therapy because of its shorter duration of action and because it can be given intramuscularly. If the response does not appear beneficial, the drug can be discontinued with the loss of blockade in two to three hours.

In the case of fulminating heart failure a continuous infusion of Arfonad is probably the therapy of choice if hexamenthonium is not adequate. Care must be taken so that excessive fluid is not administered. If ganglionic blockade appears to be adequate, one of the longer acting agents can be substituted for the hexamethonium after two or three days. This allows for less frequent administration. Mecamylamine is a particularly good substitute since, after dose adjustment, and when the patient's sensorium becomes clear, it is possible to switch to the oral route of administration employing the same dose of the drug as was effective by the parenteral route.

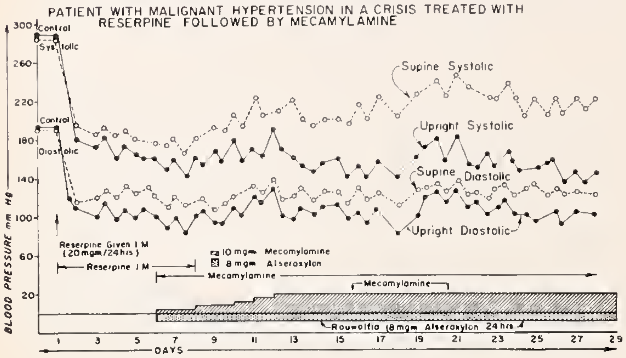


Fig. 16. Patient with malignant hypertensive encephalopathy who required parenteral Reserpine for initial reduction in blood pressure. After the blood pressure was regulated and the encephalopathic manifestations cleared, the patient was transferred slowly to a combination of Rauwolfia and a ganglionic blocking agent (Mecamylamine) administered orally. The blood pressure reduction was greatest in the standing position while receiving the latter agent. (Courtesy *A.M.A. Archives of Internal Medicine,* 98:427, 1956).

When ganglionic blocking agents given in combination with reserpine are not adequate, parenteral Veratrum should be given. Continuous intravenous infusion is the most potent and most effective approach, but the intramuscular route is preferred when full-time competent nurisng care is available, since excessive hypotensive episodes are less likely to occur following intramuscular administration.

After the hypertensive emergency is over and the blood pressure has been stabilized for three to seven days and the general status of the patient seems adequate, a permanent type program for blood pressure regulation should be substituted for the parenteral medication. A combination of Rauwolfia and a ganglionic blocking agent is usually the therapy of choice. Rauwolfia given by the oral route should be substituted for the parenteral reserpine. A dose of 1 mg. of reserpine, 250 mg. of the whole root or 4 mg. of alseroxylon, is usually given. After several months, this dose can be decreased until the smallest effective dose is being employed.

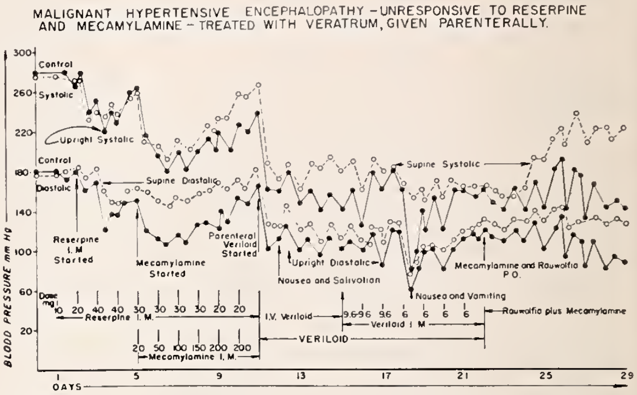


Fig. 17. Treatment of a hypertensive crisis in a patient who did not obtain an adequate response tŏ Reserpine or to ganglionic blocking agents administered parenterally. A sharp and consistent reduction occurred when Veratrum was administered intravenously, but nausea was seen. The blood pressure was controlled with parenteral Veratrum; following this the cerebral manifestations cleared up. The patient was then transferred to a combination of Rauwolfia and Mecamylamine given orally. On this combination the blood pressure reduction was primarily in the orthŏstatic position, in contrast to the response when Reserpine was given, as well as when the alkavervir (Veriloid) was given by continuous infusion. (Courtesy *A.M.A. Archives of Internal Medicine,* 98:427, 1956).

As the effect of the parenteral reserpine is lost, one of the blocking agents is started and the dose gradually increased. The dose of the blocking agent must be adjusted according to the standing blood pressure (Fig. 16). It is preferable to have the patient completely ambulatory while adjusting the drugs administered orally.

When the BUN is elevated, this determination should be repeated every two or three days while the blood pressure is being regulated. When evidence of rising blood urea nitrogen is observed, the pressure should be allowed to increase slowly by decreasing the dose of the blocking agent until the blood urea nitrogen again decreases to pretreatment levels. As the blood pressure is controlled for prolonged periods, progressive vascular deterioration is usually arrested, and renal function will usually improve if it has not progressed beyond "the point of no return" prior to therapy.

Newer Concepts in the Treatment of Hypertension

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SINCE the advent of new and more effective hypotensive drugs, it is more than ever incumbent on the physician to determine which patients need treatment and to individualize the program of treatment so that the best results may be obtained with the least side effects. Moreover, access to effective palliative treatment does not excuse the physician from seeking potentially curable causes for hypertension, such as coarctation of the aorta, pheochromocytoma, primary aldosteronism, and unilateral renal disease.

Administration of potent hypotensive drugs should be preceded by adequate evaluation of the cardiovascular and renal systems, for such data have an important bearing not only on the choice of drugs but also on the prognosis. Numerous determinations of blood pressure should be made over an extended period, because insufficient pretreatment data on blood pressure are often responsible for unnecessary, misguided or erroneously evaluated treatment.

Choice of Patients for Treatment

No magic formula exits to guide the physician in choosing the patient who needs specific hypotensive treatment. Some physicians advocate treatment for all patients with hypertension, whereas others treat only those whose hypertension is complicated or symptomatic. The introduction of effective medical treatment for hypertension has been too recent to allow this dispute to be settled with finality, but rapidly accumulating experience indicates that the proper course lies closer to the former of these divergent opinions.

Hypertension may be uncomplicated or it may be complicated by the presence of hypertensive heart failure, coronary atherosclerosis with or without myocardial infarction, cerebral vascular insufficiency with or without cerebral infarction, cerebral hemorrhage and renal insufficiency. True hypertensive headache is considered to be a symptom and not a complication of hypertensive vascular disease for, unlike the others, it apparently has no bearing on prognosis.

Uncomplicated Hypertension.—When essential hypertension is uncomplicated, the best single criterion for determining the prognosis is the appearance of the optic fundi. The prognosis is so poor for patients who manifest the findings of Group 3 or Group 4 hypertensive retinopathy[1] that vigorous hypotensive therapy is urgently indicated even in the absence of complications or symptoms.

Patients with retinal findings of Group 1 or Group 2 hypertension, without cardiovascular or renal complications, have a relatively favorable prognosis. Therefore, some physicians prefer to observe these patients, employing such measures as mild sedation, adequate rest and reduction of weight, if indicated, and withholding specific hypotensive drugs. It is logical that patients in this group who have consistently high diastolic pressures are in more urgent need of treatment than are patients whose blood pressure is more labile.

The age and sex of the patient also must be taken into consideration in deciding which patients need treatment. By and large, women tolerate hypertension better than men do. It is a clinical impression that elderly patients, especially those who have high systolic and relatively normal or only slightly increased diastolic pressures, do not often get into trouble because of the hypertension. Treatment is usually advisable for patients whose family histories reveal that hypertension has frequently led to complications and untimely death among relatives.

In the final analysis, each physician must satisfy his own conscience in deciding to what extent the patient is to be inconvenienced by treatment for

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TABLE I. PREPARATIONS OF RAUWOLFIA SERPENTINA AND THEIR DOSES

Preparation	Daily Dose (Single or Divided), mg.
Whole root (raudixin)	100-500
Alseroxylon fraction (rauwiloid, rautensin)	1.0-6.0
Reserpine (single alkaloid; preparations include serpasil, reserpoid, sandril, rau-sed, serpiloid)	0.1-1.0

a disease causing no symptoms. In arriving at this decision, the physician should ask himself two questions: (1) If he were the patient, would he want treatment? (2) Why do insurance companies "rate up" or refuse to insure hypertensive applicants, even though the hypertension is asymptomatic and uncomplicated? Furthermore, it behooves any physician who withholds hypotensive treatment from a patient to follow that patient closely, so that progression of the hypertensive vascular disease can be detected early, before it has led to irreversible complications.

Complicated Hypertension.—In the absence of overt evidence of coronary insufficiency, hypertensive heart disease manifested by significant cardiomegaly, with or without congestive heart failure, is an indication for prompt reduction in blood pressure.

It is surprising that patients whose hypertension is complicated by symptomatic coronary or cerebrovascular insufficiency usually tolerate gradual and cautious reduction of blood pressure without manifesting increased symptoms of myocardial or cerebral ischemia. Occasionally, it is advisable to start anticoagulant therapy for such patients before an attempt is made to reduce their blood pressure. Many hypertensive patients who have coronary insufficiency and angina pectoris derive considerable symptomatic improvement from treatment with hypotensive drugs. It is probably inadvisable to reduce the blood pressure of patients who have had cerebral or myocardial infarction within the previous six weeks, unless the blood pressure is extremely high and causing cardiac embarrassment. After the acute phase of the infarct has passed, hypotensive therapy is indicated.

Cerebral hemorrhage is an indication for prompt and drastic reduction of blood pressure, at times to hypotensive levels. Reduction of the blood pressure of patients whose hypertension is complicated by renal failure and azotemia often fails to improve the renal function; indeed, at times it appears to impair it further. However, the prognosis is so uniformly poor when azotemia supervenes that little is to be lost by a cautious trial of treatment.

Choice of Drugs

With the bewildering array of proprietary preparations, including confusing combinations of drugs in one tablet, now being marketed for the treatment of hypertension, it is well to remember that only four classes of drugs, exclusive of sedatives and tranquilizers, are of sufficient importance to warrant discussion. The specific hypotensive drugs to be discussed always should be administered in conjunction with, and not in lieu of, the less specific but time-honored and proved measures of reduction of weight, rest, relaxation, reassurance and sedation.

The choice of drugs and their doses must be individualized to meet the needs of each patient, for it is perilous to treat hypertension by stereotyped rules. For this reason, the use of tablets containing combinations of drugs is to be deprecated.

Rauwolfia.—An adequate trial of one of the derivatives of Rauwolfia serpentina is justified in most cases of hypertension when specific therapy is indicated. Since its hypotensive effect is neither drastic nor prompt, this agent should not be relied on as the sole hypotensive drug when prompt reduction in blood pressure is compulsory, unless it is given in relatively large parenteral doses (3 to 5 mg.). Doses of this size should not be administered for more than a few days. There is no evidence that any one preparation of Rauwolfia is superior to others (Table I). Side effects include nasal stuffiness, drowsiness, bradycardia, mild laxation, weird and bizarre dreams, Parkinsonian-like rigidity and mental depression. Only the last two effects are of such serious import as to compel prompt cessation of treatment. A previous history of mental depression or even a tendency toward mental depression constitutes a relative contraindication to the use of Rauwolfia, for major depression requiring electroshock therapy has been induced by these preparations. Rauwolfia should be administered cautiously, if at all, in the presence of an active peptic ulcer, since it increases gastric acidity.

Hydralazine (Apresoline) Hydrochloride.—Hydralazine hydrochloride produces a high incidence of unpleasant side reactions, including headache, nausea, vomiting, flushing, tachycardia, peripheral edema, fever and arthralgia. For this reason, it is seldom used alone, but it is excellent in combina-

TABLE II. PREPARATIONS OF VERATRUM AND RECOMMENDED DOSES

Preparation	Daily Total Dose (Usually Divided into Four Doses)
Whole powdered Veratrum viride (vertavis)	30-90 Craw units
Extract of ester alkaloids of Veratrum viride, such as alkavervir (veriloid) and cryptenamine tannate (unitensen)	8-24 mg.
Purified alkaloids of Veratrum alba, such as protoveratrines A and B (veralba) and protoveratrine A and B maleates (provell maleate)	0.8-3.0 mg.

TABLE III. GANGLION-BLOCKING AGENTS AND DOSES

Drug	Single Dose, mg.	Doses per 24 Hours
Hexamethonium chloride (methium, hexameton)	125-1,500	4 or more
Pentolinium tartrate (ansolysen)	20-500	3-4
Chlorisondamine chloride (ecolid)	12.5-200	2-3
Mecamylamine hydrochloride (inversine)	2.5-30	2-3

tion with Rauwolfia when hypertension cannot be adequately controlled with Rauwolfia alone. It also can be used advantageously in combination with a ganglion-blocking drug in the treatment of severe hypertension. Unfortunately, many physicians have been alarmed by reports of a collagen or "lupus" type of reaction resulting from prolonged administration of hydralazine in large doses. Clinically, this reaction resembles disseminated lupus erythematosus, and L.E. cells have been found in the blood of patients with this syndrome. The symptoms subside when the drug is withdrawn, and this reaction infrequently occurs when the daily dose of hydralazine does not exceed 200 mg. The usual single dose of hydralazine is 25 or 50 mg. given three or four times a day.

Veratrum (Table II).—All preparations of Veratrum now available share the glaring disadvantage of an extremely narrow margin between therapeutic and toxic doses. When Veratrum is used alone in the treatment of hypertension, toxic effects may appear before a therapeutically effective dose has been reached. The toxicity of Veratrum is unpleasant, although seldom dangerous. It consists of crises characterized by bradycardia, salivation, hiccup and heartburn, often followed by nausea and vomiting. Hypotension is an inconstant feature of a crisis induced by use of Veratrum. Consequently, it is advantageous to use Veratrum in combination with Rauwolfia or hydralazine or both, because it is often possible to obtain adequate reduction of the blood pressure with a minimum of side effects by using small doses of two or more drugs.

Ganglion-Blocking Drugs (Table III).—Unequivocally, the most potent hypotensive drugs available today belong to the group of ganglion-blocking agents. Because of their potency and side effects, administration of these drugs is not without danger and requires expert supervision by the physician, as well as the careful and often tedious cooperation of the patient. For these reasons, treatment with ganglion-blocking agents should be reserved for those patients in whom it is compulsory that hypertension be controlled promptly. These include patients with Group 3 or Group 4 hypertensive retinopathy and patients whose hypertensive vascular disease has progressed in spite of treatment with combinations of less potent drugs in adequate doses. Because of their specific action in reducing central venous pressure, the ganglion-blocking agents are especially helpful when hypertensive heart disease has led to congestive heart failure.

It is usually advisable that treatment be instituted and dosage adjusted while the patient is hospitalized. Treatment is started with the smaller doses shown in Table III, and each dose is changed by increments equal to the initial dose every second or third day until the blood pressure is satisfactorily controlled or until side effects preclude further increments. Certain patients may tolerate effective doses of one drug better than another, but this can be determined only by trial. It is preferable to give ganglion-blocking drugs at regular intervals, usually every six, eight or twelve hours, depending on the response of the blood pressure.

Experience has shown that treatment with ganglion-blocking agents is most effective when the patient or a member of his family or both have been instructed in the technique of measuring blood pressure, since in most cases the blood pressure must be used as a guide to make appropriate modifications in doses from day to day. For instance, patients are instructed to omit the ganglion-blocking agent whenever the systolic blood pressure is less than 120 mm. of mercury at the designated time for a dose. When the systolic blood pressure is between 120 and 140 mm. of mercury, only half the dose is taken. This is analogous to the situation in which a diabetic patient tests his urine for sugar as a guide

TABLE IV. PHARMACODYNAMICS OF GANGLION-BLOCKING AGENTS

Site of Block	Observed Effects	Antidote or Treatment
Sympathetic ganglia	Orthostatic hypotension	Reduce dosage; recumbent position
	Excessive hypotension	Elevate foot of bed; pressor drugs (neo-synephrine, levarterenol)
Parasympathetic ganglia	Blurred vision	Pilocarpine eye drops; use of a positive lens
	Oral dryness	Pilocarpine nitrate (5 mg.) with each dose; chewing gum
	Constipation	Laxatives; prostigmin bromide (15 mg. sublingually or orally) as needed
	Paralytic ileus	Stop use of drug; prostigmin methylsulfate (1.0 mg. IM); intubation
	Urinary retention (older men)	Prostigmin bromide (15 mg. sublingually or orally); urecholine chloride (5 to 10 mg. orally); void before each dose; prostatic resection
	Impotence	Omit occasional dose

to dosage of insulin. To eliminate as many variables as possible, patients are instructed to take their blood pressures while sitting and to use the same arm each time.

Mecamylamine (inversine) hydrochloride is the only drug of this group that is absorbed completely from the gastrointestinal tract. This accounts for the rather small dose of this preparation compared to that of the others.

Drugs of this group reduce blood pressure by blockade of the autonomic ganglia of the sympathetic nervous system. Unfortunately, the blocking action of these drugs on the parasympathetic ganglia is also evident, and this gives rise to most of the undesirable and untoward side effects (Table IV). Patients can be reassured that many of these side effects diminish or disappear with prolonged use of the ganglion-blocking agent. Suggestions to alleviate some of these side effects are presented in Table IV. The number of patients who must abandon use of these drugs because of side effects bears an inverse relationship to the time which the understanding and sympathetic physician is willing to take to encourage his patients during the first few weeks of treatment. Special care must be taken to insure that patients who receive ganglion-blocking drugs have regular bowel movements, since paralytic ileus is a dangerous and occasionally fatal complication of treatment with these agents. Mecamylamine is the only one of this group of ganglion-blocking drugs that has been reported to produce a bizarre coarse tremor and psychosis. Hexamethonium is the only one of the group that has produced fatal interstitial pneumonitis. When these rare side effects occur, the patients so affected almost always are azotemic.

The concomitant use of Rauwolfia or apresoline or both with ganglion-blocking agents sometimes makes it possible to achieve smooth and effective reduction in blood pressure with smaller doses of the ganglion-blocking drug. This will eliminate or ameliorate some of the side effects.

Sympathectomy for Hypertension

Lumbodorsal sympathectomy usually is reserved for those patients whose blood pressure cannot be controlled by adequate doses of hypotensive drugs or whose temperament is such that they cannot tolerate or will not adhere to a strict medical regimen for the treatment of their hypertension.

Objectives of Treatment

The ultimate objective of the treatment of hypertension is to prevent or forestall the complications that cripple or kill. In the light of present knowledge, it appears that this can be accomplished best by reducing the blood pressure to normal or to as nearly normal a level as the patient can tolerate. The best immediate criterion, then, for the success or failure of treatment is the response of the blood pressure. Treatment should be pursued until the blood pressure is brought under satisfactory control or until side effects from the drugs preclude further increment in dosage. The limits of doses given in the tables are arbitrary and may be exceeded if necessary when the condition is resistant. If the blood pressure is not reduced, treatment is a failure and should be either abandoned or modified to produce the desired effect, for, so far as is now known, there is no virtue in administering ineffective doses of hypotensive drugs.

Results of Treatment

When the proper drugs or combinations of drugs are administered in adequate doses, blood pressure can be reduced significantly in most patients with hypertension. Reduction of blood pressure so achieved often produces astounding improvement in cardiac function and hypertensive retinopathy. Cardiac enlargement recedes, electrocardiographic evidence of left ventricular strain may disappear, signs and symptoms of congestive heart failure abate, and angina pectoris may improve remarkably. The retinopathy of malignant hypertension regresses with regularity. Hypertensive headache usually diminishes in severity or disappears entirely. The lives of many patients with Group 3 or Group 4 hypertensive retinopathy undoubtedly have been prolonged by adequate medical treatment.

However, insufficient time has elapsed since the advent of effective hypotensive treatment to permit any conclusions with regard to the prolongation of life for patients with the retinal findings of Group 1 or Group 2 hypertension.

It is discouragingly clear that many patients whose hypertension is adequately controlled continue to suffer from, and all too frequently succumb to, those complications of hypertension that arise from coexisting atherosclerosis, namely cerebral and myocardial infarction. It is reasonable to assume, of course, that such complications result from extensive cerebral or coronary atherosclerosis that was present before blood pressure was controlled. More time must elapse and more patients must be treated during the early phase of their hypertension to determine whether adequate control of blood pressure will indeed retard atherogenesis.

Most disappointing is the failure of hypotensive therapy to influence beneficially the course of renal insufficiency, once this complication has occurred.

Disadvantages of Present Drug Therapy

Drug therapy of hypertension, although more effective now than ever before, is expensive, time-consuming for both patient and physician, and fraught with many unpleasant side effects, some of which are serious. Moreover, treatment is palliative and not curative, implying that it is a lifelong proposition. Physicians become dismayed when patients abandon an otherwise successful regimen because it is tedious and unpleasant. But discontent on the part of physicians is good, for it keeps them constantly searching for a more convenient, a more effective and a safer way to deal with the omnipresent problem of hypertension.

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PRESSOR AMINES IN PHEOCHROMOCYTOMA

(Continued from Page 296)

was not found. After operation, the concentration of epinephrine-like substance was 2.6 and 1.9 micrograms per liter of plasma five and seven days, respectively, after the last use of nebulized epinephrine. At the time these blood samples were obtained after operation, results of the regitine tests were also normal. It was thought that absorption of epinephrine from the pulmonary parenchyma may have been sufficiently slow and the excretion impaired enough, as evidenced by a pre-operative blood urea of 70 mg. per 100 ml., to result in these increased concentrations of pressor amines.

A number of therapeutic agents undoubtedly may interfere with the chemical or biological assay of pressor amines, or with both techniques. Only with constant vigilance and by repeatedly investigating the effects of various therapeutic agents *in vitro* and *in vivo* on the fluorometric method of analysis and on the plasma concentrations of epinephrine and norepinephrine can one avoid drawing erroneous conclusions from the results of such fluorometric quantitation.

It was found essential in one of the patients with sustained hypertension and two of the patients with paroxysmal hypertension to obtain blood during or shortly after a paroxysm of hypertension, either naturally occurring or induced by anesthesia or a drug such as histamine, in order to increase the pressor amines to a concentration suggesting a diagnosis of pheochromocytoma. The percentage of norepinephrine in the plasma obtained pre-operatively was greater than that of epinephrine in all but three of the afore-mentioned twenty-three patients. This finding is consistent with the greater concentrations of norepinephrine than of epinephrine noted in the majority of tumor extracts. However, no close correlation was apparent between the percentage of pressor amines in the plasma and that in the tumors.

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Segmental Occlusion of the Femoral Artery and Femoral and Popliteal Aneurysms

Surgical Aspects

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THE MAJORITY of patients presenting themselves with the complaint of intermittent claudication of the lower extremities could be afforded little, if any, relief until recently. While lumbar sympathectomy has given some relief of symptoms due to ischemia and has often prolonged the life of extremities, it has only infrequently provided relief from intermittent claudication.

The more recent direct surgical approaches (restoring direct continuity of blood flow or with by-pass technique) have resulted in restored muscle function to the point of relief from intermittent claudication. These newer methods have also resulted in healing of gangrenous areas in the more ischemic limbs or, in many instances, allowing for a lower level of amputation than might otherwise have been possible. In the case of occluding arterial lesions, the indications for surgery often are incapacitating claudication, if not other manifesations of ischemia. The possible loss of a limb seems to be a readily accepted risk in the patients with severe ischemia.

When an asymptomatic aneurysm of either the femoral or popliteal arterial vessels is found, the indications for surgery somehow seem to be less urgent, both to the patient and often to the physician, as well.

The report of Gifford, Hines, and Janes,[1] on the analysis and follow-up of 100 popliteal aneurysms, showed that twenty of these developed an acute thrombosis, sixteen ruptured, fourteen were responsible for distal emboli and twenty-four were the cause of a distal gangrene.

Even after thrombosis, repeated distal embolism, episodes of leakage and in spite of impaired function and of the obvious threat of recurrence with more serious consequences, the patient and often his physician, has hesitated to allow surgical treatment. The question heretofore has always been whether the surgical treatment might not make matters worse.

The aneurysmorrhaphy procedure of Matas,[2] which he performed in 154 instances, reduced the incidence of gangrene, in his series, from 10 per cent to 5.2 per cent. This report, however encouraging, did little to popularize surgical treatment of arterial aneurysms of the lower extremity.

In 1946, Lilly[3] proposed the procedure of lumbar sympathectomy followed by endaneurysmorrhapy. In three of his four cases he did a chemical sympathectomy using alcohol injections with good results. In the fourth patient he extirpated a ruptured aneurysm, after lumbar sympathectomy, also with a good result. It became apparent then, that one might perform a lumbar sympathectomy on patients with popliteal aneurysm and shortly therafter remove the aneurysm with relative impunity.

Stimulated by this, Linton[4] resected popliteal aneurysms in fourteen patients after preliminary sympathectomy without the loss of any limbs. This method, leaves the patient with a viable extremity, free from the threat of aneurymal rupture, embolism and thrombosis as brought out again in the detailed report of Gifford, Hines and Janes.[1] However safe, most of these limbs have impairment in function, in the form of intermittent claudication, as revealed by Julian and his associates.[5] This then, puts these limbs into the category with other arteriosclerotic lesions, in which restoration of adequate arterial blood flow is the ideal to be attained.

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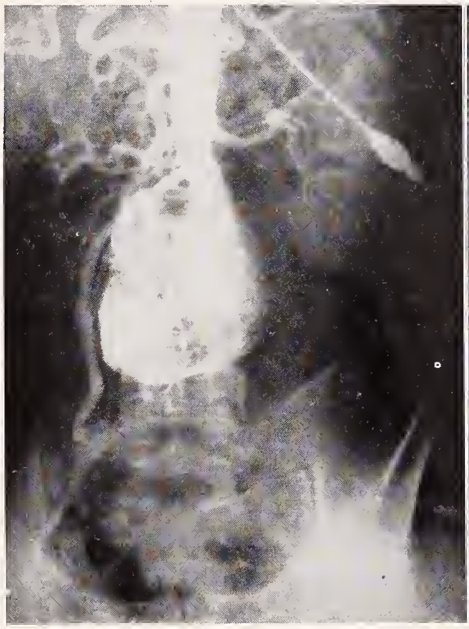

Fig. 1. *(left)* Aortogram in a forty-two-year-old white man made nine months after wiring procedure and aortoiliac shunt with an autogenous saphenous vein.

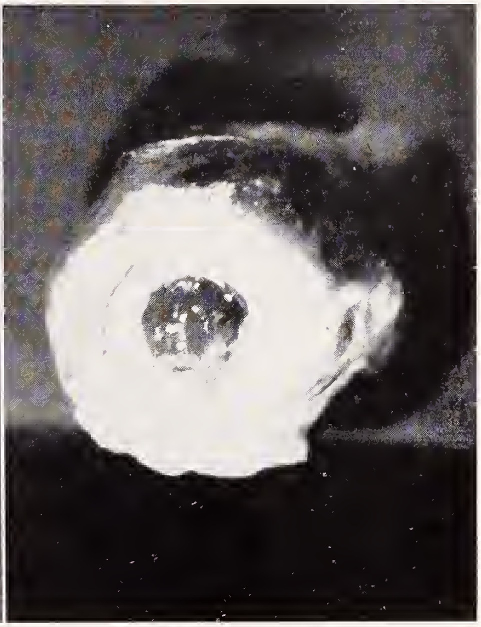

Fig. 2. *(right)* Thrombosed ilio-femoral Ivalon prosthesis removed two years after implantation for ruptured aneurysm of the common femoral artery. Microscopic section of this vessel showed marked proliferative connective tissue reaction with no signs of fibroblastic ingrowth.

Failures and disappointments in this country, based on the experience of those who have attempted extensive thromboendarterectomy and resections and replacement of long occluded femoral arterial segments have already been documented.[6,7] On the other hand, Cannon and Barker[8] have reported encouraging results in twenty out of twenty-three patients with extensive femoral occlusions after thromboendarterectomy, in instances where the popliteal and more distal vessels were patent. Warren,[9] using the same technique, however, was disappointed with the results in this type of lesion.

Although restoration of continuity of blood flow, using a vein graft, is not new,[10] it had certainly not been tried extensively until the report of Kunlin of Paris, in 1951.[11] We successfully used a saphenous vein to by-pass an aortic aneurysm in 1951, that had been wired previously (Fig. 1). This remained open until the patient's death, at approximately eighteen months postoperatively, which presumably resulted from a rupture of his abdominal aneurysm.

The early report of Julian, et al,[6] of femoral artery replacements using veins, was encouraging and the later reports of more extensive work using arterial homografts, both as direct grafts and by-passes (as in the series reported by Crawford, et al,[12]) have made the restoration of continuity of flow a practical reality. It has also been learned from experience to date, that a prime factor in the success of either endarterectomy or grafting is the presence of an adequate patent peripheral arterial tree or "run off."

The short supply of prepared or fresh arterial homografts stimulated the search for other prosthetics. Of the various materials being used at the present time, our own experiences, aside from the use of veins, has been mainly with the use of Ivalon compressed sponge, originally recommended as vascular prostheses by Shumway and his co-workers[13] and the braided nylon tubes originally described by Edwards and Tapp.[14]

Experiences in the Surgical Management of Obliterative Arterial Lesions in the Lower Extremity

Thromboendarterectory.—Using direct exposure and multiple extensive longitudinal arteriotomies, as described by Dos Santos[15], thromboendarterectomies were performed in three femoral arteries, from external iliac to popliteal level. One patient expired on the tenth postoperative day of a myocardial infarction, after extensive hemorrhage from the operative site and in one patient the entire area thrombosed within a few days and required

TABLE I. ILIAC—FEMORAL ARTERIAL SHUNTS OR REPLACEMENTS

Results in 24 cases

Prosthesis	Number	Success	Failure
Autogenous saphenous vein	8 (2*)	7	1
Nylon (Edwards-Tapp)	5 (1*)	5	0
Ivalon	11 (3*)	5	6 (3*)
Total	24 (6*)	17	7

*Replacements

TABLE II. FEMORAL—POPLITEAL ARTERIAL SHUNTS OR REPLACEMENTS

Results in 73 cases

Prosthesis	Number	Success	Failure	Expired
Saphenous vein	37 (1*)	29 (5-24 Mos.)	8 (1*)	
Nylon (Edwards-Tapp)	32	16 (3-12 Mos.)	13	3
Ivalon	4	0	4 (1 Mo.)	
Total	73	45	25	3

*Replacements

a mid-thigh amputation. The remaining patient had a good result with relief of claudication and has a persistent peripheral pulse after three years.

Short segment thromboendarterectomies were performed in five patients with acute arterial occlusions. Two of these were due to embolism and two to thrombosis. All were seen after twelve hours from the acute onset and three were at least twenty-four hours after the acute onset. One of the patients seen after twenty-four hours expired within twenty-four hours of surgery and was found to have multiple emboli in the renal, splenic and cerebral vessels. Another patient seen twenty-four hours after onset, required a saphenous vein graft of the common femoral artery, since the remaining vessel, in the site of the embolus, after endarterectomy was too friable to hold sutures. This patient is well, with good peripheral pulses, thirty months after surgery. The remaining three patients had patent vessels on follow-up examination, two at one year and one at three months.

Iliac Femoral Arterial Replacements and Shunts.—The results of our twenty-four iliac-femoral arterial shunts and replacements are summarized in Table I:

Of the eight instances in which an autogenous saphenous vein was used, there was one failure and this was in a shunt with poor distal "run-off" causing a thrombosis in the immediate postoperative period. The successful vein grafts have all existed over two years. The five Edwards-Tapp nylon tubes used at this level, have remained open for from three to twelve months. Tubes of 5/16 inch diameter were used in all instances.

Of the eleven compressed Ivalon prostheses used at this level, six failed. Three of these were direct replacements. One became thrombosed within the first twenty-four hours after surgery, one at three months and one after two years. Of the three shunts with Ivalon that failed, all were thrombosed within twenty-four hours of the surgery. The successful Ivalon grafts were all patent over fourteen months, at the last follow-up.

Of this latter group, only four patients had replacements for femoral aneurysms, all of which were either leaking or had ruptured.

Femoral Aneurysms.—A total of nine femoral aneurysms were seen in this series of patients. Two of these were ruptured arteriosclerotic lesions of the common femoral artery. When first seen, and were replaced by Ivalon prostheses. One of these became infected, thrombosed and was removed at two months. This patient expired and is included in Table II. The other became thrombosed at two years and was removed (Fig. 2). This was replaced by a braided nylon tube and is included as a successful graft in Table I.

Two traumatic aneurysms of the superficial femoral artery, were replaced by Ivalon prostheses and both failed, due to hemorrhage, secondary to postoperative heparinization and had to be removed. Both limbs survived with functional ischemia. These are included as failures in Table II.

Four other aneurysm lesions of the superficial femoral artery were treated by lumbar sympathectomy, alone, and one by wrapping with Ivalon. All were free of symptoms from either embolism or thrombosis after three years.

Femoral-Popliteal Arterial Shunts.—Table II summarizes the results in this group of seventy-three instances of surgery.

Twenty-nine out of thirty-seven saphenous, autogenous and homogenous venous shunts or replacements were open at follow-up examination, five to twenty-four months after surgery.

In one instance, an autogenous vein was used to bridge a traumatic occlusion. This thrombosed within a few hours. In at least two instances, the shunts used were composed of both the patient's own saphenous vein and one or more segments of homogenous vein grafts.

Sixteen of thirty-two Edwards-Tapp nylon shunts were still open at from three to twelve months' follow-up examination. Thirteen of these shunts failed. Two of these failures were instances in which a prosthesis of ¼ inch diameter was used. An almost immediate thrombosis occurred in each instance. The remaining failures were associated with poor run-off and with thrombosis of the shunt within two months of the operative procedure.

In one case (mentioned above under Femoral Aneurysms), there was a frank infection along the course of the prosthesis and the patient succumbed to a septicemia. In two other cases, patients died in the immediate postoperative period of coronary arterial disease.

Compressed Ivalon prostheses were used in four instances and failed in all. In two patients, almost immediate thrombosis resulted and in two (mentioned above under Femoral Aneurysms), repeated hemorrhage through the graft, secondary to postoperative heparinization, necessitated finally, ligation of the femoral artery.

Popliteal Aneurysms.—In Table III, are summarized our results with twenty popliteal aneurysms.

Eleven popliteal enurysms were treated with lumbar sympathectomy. Eight out of these eleven were thrombosed at the first onset of symptoms. Of this group, four patients had repeated peripheral embolic episodes within one year. One of these produced gangrene and required amputation. One of the group ruptured and required subsequent resection. Three of these original eleven had subsequent resection of the aneurysm. One patient in whom a primary lumbar sympathectomy and resection of the aneurysm were done, died in the first week postoperative, of acute coronary insufficiency.

One patient was seen for the first time with a ruptured aneurysm and impending gangrene of the extremity. Because of his poor general condition, a simple resection of the aneurysm was done, yielding a good result—a usable extremity but with claudication.

In eight instances, primary resection with autogenous vein grafting was attempted, after sympathectomy. Four of these failed almost immediately, due to thrombosis and resulted in viable extremities but with claudication. Four were successful and have functionally good extremities without claudication.

TABLE III. SURGICAL TREATMENT OF POPLITEAL ANEURYSMS

Results in 20 cases

Treatment	No.	Success Functional	Claudication	Amputation	Expired
Lumbar sympathectomy	11		10	1	0
Sympathectomy & resection	(3)		(3)		(1)
Resection only	1		1		
Sympathectomy resection and vein replacement	8	4	4		
Total	20	4	15	1	(1)

Discussion

Encouraged by the experimental work of Shumway[13] and by our own successful use of compressed Ivalon sponge prostheses in the replacement of aortic lesions, we attempted its use at the iliac and femoral level. As had recently been documented experimentally by McCaughan[16] we found that replacement of vessels (with this material) having an inner diameter of 8 mm or less, would result in failure from early thrombosis in a high percentage of instances. We now have begun to see late failures from thrombosis of iliac and femoral Ivalon prostheses. As can be seen in Figure 2, there is a great deal of reaction surrounding this material, making it a thick, firm tube in contrast to the pliable and elastic one that was originally inserted. This makes accommodation to motion practically impossible.

With motion, as in the hip joint, the host vessel may become occluded by folding against the end of this mass and this cause occlusion and subsequent thrombosis. This may have been the mechanism of occlusion of the graft shown in Figure 2. In view of these experiences, we have abandoned the use of compressed Ivalon tube prostheses in areas distal to the common iliac or similar-sized vessels where motion may be a problem.

It seems best to leave a patient with his own blood vessels, if they can be repaired and will heal to a reasonably good state of function. Thromboendartectomy, therefore, seems to be a procedure of choice.

In spite of the fact that we have not used it extensively, we are interested in the further developments of the method which might give more promise of a lasting functional result.

For short segments and especially in instances of acute occlusion, either by embolism or spontaneous thrombosis in severely sclerotic femoral arteries, we feel that endarterectomy is definitely necessary for a successful result.

Our experience with the treatment of long-segment femoral arterial occlusions has shown the by-pass technique (using autogenous saphenous vein or crimped braided nylon tubes—Tapp-Edwards, 5/16 inch diameter) to be most successful. However, the follow-up of patients, in whom saphenous veins have been used, is only a little over two years and those in whom braided nylon tubes have been used, only one year. This limits our drawing any final conclusions on the relative merits of each method.

The most satisfactory handling of the symptomatic or asymptomatic popliteal eneurysm, appears to be sympathectomy followed by immediate resection. Since the incidence of functional ischemia (intermittent claudication) is so great in these patients, an attempt at restoration of continuity of blood flow should be made using either an autogenous vein or crimped nylon prosthesis and employing an end-to-side technique for the distal anastomosis.

The most satisfactory distal vascular anastomosis in arteriosclerotic occlusive disease, at this time, appears to be that made with a by-pass, end-to-side technique into the proximal popliteal artery, as described by Linton.[7] However, the technique of McAllister[17]—that of leaving the diseased vessel in place and making an end-to-end distal anastomosis is worthy of further trial. In the replacement of popliteal aneurysms we have had only four out of eight successful procedures with autogenous veins using the end-to-side technique. Not only does the diseased condition of the distal vessels affect the end results here but also implicated is the small calibre of the vessels involved. The problem at this level is similar to that encountered in the surgical approach to coronary artery disease. It seems that for vessels of such small calibre, replacement is almost a practical impossibility but some types of thromboendarterectomy, such as by hydraulic dissection instead of mechanical dissection, may hold more promise.

We believe that for successful vascular reconstructions, certain of the principles stated by Linton[7] should be adhered to, namely:

1. A suitable graft must be available.
2. The site of proximal anastomosis must be high enough in the patient's arterial system to supply an ample volume of blood at an effective pressure.
3. There must be an adequate distal "run-off."
4. An arteriogram should be made pre-operatively to determine if the above conditions (2 and 3) are satisfied.
5. Meticulous technique should be used and excessive trauma avoided.
6. Dilute Heparin should be used in occluded distal vessels and in the operative site—(0.02 per cent) solution during anastomosis but not post-operatively.
7. Most rigid asepsis should be practiced.

Summary

The results of experiences are presented in twenty-four iliac-femoral arterial shunts or replacements and seventy-three femoral-popliteal arterial shunts or replacements using autogenous and homogenous veins, braided nylon (Edwards-Tapp) tubes and compressed Ivalon tubes. The results of our experiences in treating twenty popliteal and femoral aneurysms is also presented.

The limited experience in our use of thromboendarterectomy is also discussed.

Our most satisfactory results, for long segmental occlusions in the iliac-femoral and femoral-popliteal areas, have been with the use of autogenous saphenous veins and braided nylon tubes, both as shunts and as replacements.

Sympathectomy and resection of popliteal aneurysms has been the most satisfactory in this group of cases. The further attempts at replacement with autogenous veins, after resection of popliteal aneurysms, is described in eight cases with encouraging results in one half of these.

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Surgical Considerations in the Management of Cerebrovascular Disease

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AMONG 200 patients entering the hospital with a diagnosis of "stroke," occlusive disease was present in a little over half of the cases (Table I). In Table II are tabulated the patients with the provisional diagnosis of stroke, who proved to have surgical mass lesions. About 10 per cent of the group of 200 patients, therefore, had mass lesions needing surgical intervention. Massive hematomas are not very common among this group. In Table III are shown the diagnoses, after careful study, of certain cases with the initial provisional diagnosis of stroke.

In many instances, as seen in Table I, the pathologic disease associated with a stroke syndrome is in the neck or at the base of the brain with occlusion of the carotid or vertebral complex and the larger vessels of the circle of Willis.

Carotid artery thrombosis in the neck may be complete or partial. The occlusion may begin at the bifurcation and extend up toward the siphon, or there may be a retrograde thrombosis with the initial thrombotic involvement at the siphon. However, the greatest majority are examples of thrombosis at the bifurcation in the neck due to atheromatous changes. A complete occlusion may be fresh or chronic. In some instances an occlusion of the internal carotid artery may occur with few symptoms and signs and the disease process may be found in the routine study of the patient.

Among a little over 100 cases of complete occlusion of the internal carotid artery in the neck, a hemiplegia or hemiparesis was present in 90 per cent; there was a history of a single involvement in 70 per cent of this group; and among 30 per cent, there were multiple episodes of weakness or paralysis. The time of onset of the weakness or paralysis was during rest or in bed in 20 per cent, and during activity or daytime in about 80 per cent. The onset was abrupt in about 80 per cent.

TABLE I. OCCLUSIVE DISEASE

Carotid artery (complete)	21
Carotid artery (partial)	20
Anterior cerebral	15
Basilar artery (partial)	15
Middle cerebral trunk	9
Basilar artery (complete)	5
Post. cerebral artery occlusion	2
Arteriolar disease	32

TABLE II. SURGICAL MASS LESION

Brain tumor	16
Intracerebral hematoma	5
Subdural hematoma	3
Brain abscess	1

TABLE III. OTHERS

Embolism	11
Cerebral hemorrhage (hemorrhagic infarction)	8
Cerebrovascular insufficiency	9
Saccular aneurysm (1 A/V)	4
Cerebral atrophy	2
Post ictal state	1
Syphilis	1
Encephalitis	4
Miscellaneous	16

Unconsciousness and coma was noted in 20 per cent. Generalized or Jacksonian seizures occurred in 10 per cent; with the Jacksonian and the generalized seizures in equal numbers. Headache was present in one of four or five patients. Visual disturbances were noted in 10 per cent with homonomous hemianopic defects in half of the cases and a complete blindness of one eye in the other half. When there is unilateral blindness, the possibility of occlusion of the common, internal and external carotid arteries is much more likely.

With partial occlusions due to atheromatous plaques, the symptomatology has involved episodic

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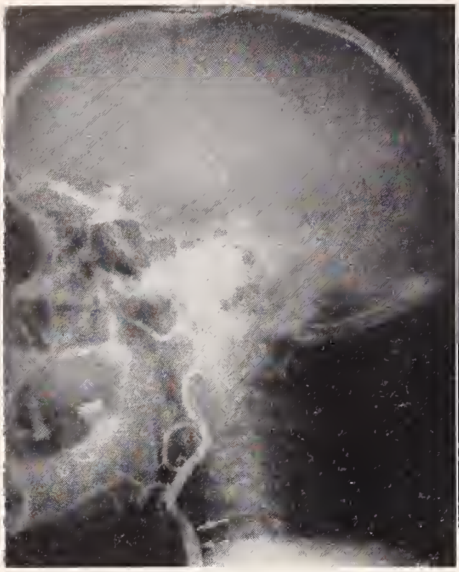

Fig. 1. Sudden onset of paralysis of the left half of the body with clear cerebrospinal fluid. Angiography showed complete occlusion of the internal carotid artery at the bifurcation. Arrows indicate excision of a portion of the internal carotid artery and cervical sympathectomy. Weakness of the left half of the body completely disappeared, patient (present age, seventy-three years) has been followed for two years with no evidence of recurrences, no anticoagulation used.

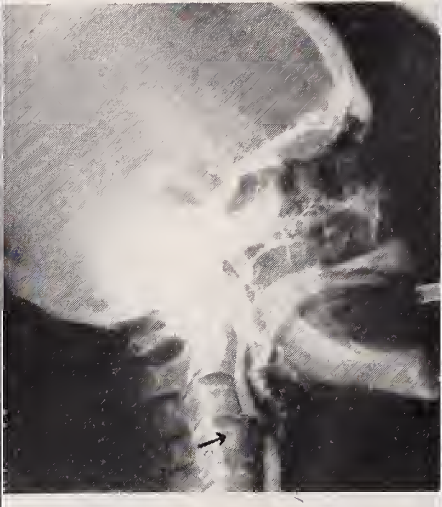

Fig. 2. Partial occlusion of the internal carotid artery at the bifurcation due to atheroma associated with repeated attacks of weakness of the left half of the body.

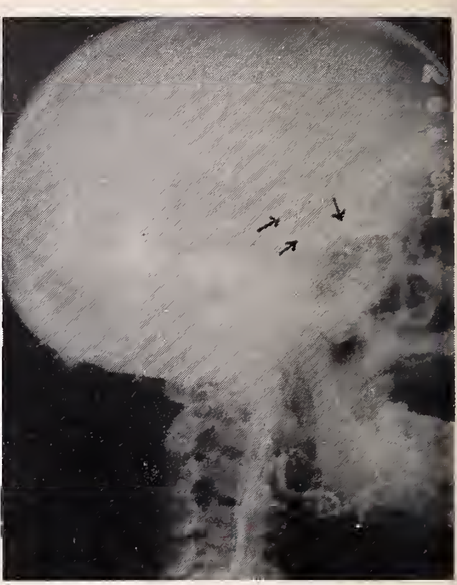

Fig. 3. Example of internal carotid artery thrombosis with visualization of a portion of the carotid siphon intracranially through collateral supply from frontal and ophthalmic vessels. Arrows point to the internal carotid siphon and the ophthalmic artery. External carotid appears to contribute significantly to the internal carotid circulation through intercommunications.

complaints of weakness or numbness in one-half or a portion of one-half of the body. In an occasional case, a more enduring weakness or paralysis has been seen. In angiograms obtained to include the carotid bifurcation, partial occlusion of the internal carotid artery is a common finding (10 per cent) among patients with "stroke" syndrome. The patients with partial occlusion may be helped by medical and/or surgical management.

Since, in many instances, atheromatous plaques may precede the eventual occlusion of the carotid bifurcation, the possibility of early diagnosis and surgical or medical management to prevent an on-coming complete occlusion must be considered.

Surgical treatment will depend upon the pathologic state revealed by angiography and exploration of the carotid artery. The cases may be divided into the following types:

1. An acute fresh occlusion in which a thrombectomy may be followed by re-establishment of the blood flow.

2. A chronic occlusion in which excision of a portion of the carotid artery and a servical sympathectomy may be of some value. It is now apparent that the external carotid circulation contributes significantly to the internal carotid circulation in cases in which the internal carotid in the neck is thrombosed. The use of sympathectomy to enhance the circulation by vasodilation of the extra-cranial vessels may be of value. Among the 25 per cent of the patients who appear to recover completely following a chronic occlusion of the internal carotid artery, the external carotid's contribution to the cerebral circulation is undoubtedly a factor.

3. A partial occlusion (and occasionally in chronic, and apparently complete occlusion) in which intimectomy may re-establish the blood flow.

Among fifty-five patients in our group, the neck was explored. In all but five, there was a chronic complete occlusion of the internal carotid. Among the five with acute occlusions, only three bled from the distal end of the artery after removal of the clot. Although the circulation was re-established acutely, postoperative angiograms performed several weeks later, revealed that in two cases, clotting had reoccurred with complete occlusion of the vessel.

When there is good bleeding from the distal end of the carotid artery and when the bifurcation is occluded by atheromatous disease, several methods of management are possible: (1) an external carotid artery shunt to the distal end of the internal carotid; (2) the use of an inanimate tube for end to end or side to end anastomosis; (3) the use of a homologous graft, and (4) intimectomy.

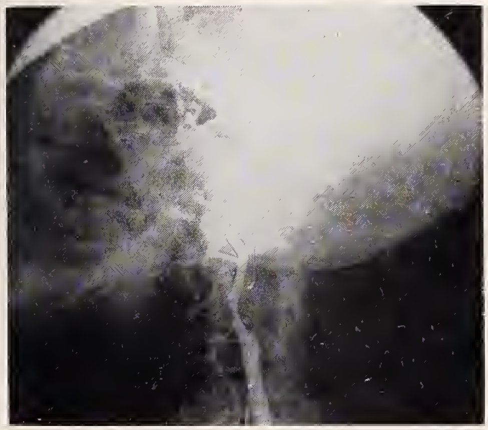

Fig. 4. A man aged sixty-three, seen six months after onset of right hemiparesis and aphasia. Angiogram revealed thrombosis of the internal carotid artery. Follow-up for two years with no improvement in the aphasia, some improvement in the weakness of the right half of the body. No operative intervention performed in this case.

Grafting of the external carotid artery to the patent internal carotid is not desirable in the authors' judgement because this destroys a good source of collateral circulation to the internal carotid through the frontal and ophthalmic arteries. The use of an end-to-end anastomosis of the common carotid to the patent internal carotid is mechanically impossible in many instances because of insufficient length. The use of end-to-end or side-to-side homografts has been satisfactory in the peripheral circulation, particularly in the lower extremities. In our hands, this method has not been satisfactory, thus far, thrombosis occurring rather rapidly due most probably to the small caliber of the internal carotid artery.

Crimped nylon grafts were investigated by trials within the aortas of dogs; this vessel is larger in size than the internal carotid artery in the adult human. In twenty-eight experiments it was found that in four cases the lumen of the tube was patent for one to three months, but in six instances, complete occlusion occurred from an intimal growth in three to seven months. Use of an inanimate tube for vessels of the size of the internal carotid artery may not be satisfactory for long periods. This is not to say that such tubes are not desirable or even ideal if the lumina remain patent indefinitely or if the temporary patency will allow sufficient time for collateral circulation to develop. More work should be done for the purpose of obtaining the best material for grafting. We have not employed nylon tubes in the human carotid thus far. In three dogs, the intima of the descending aorta was excised with the electrocautery current. The aneurysmal-like dilatation of the vessel did not progress. Later, the local dilatation became reinforced by connective tissue, the lumen of the vessel remained smooth-lined and patent.

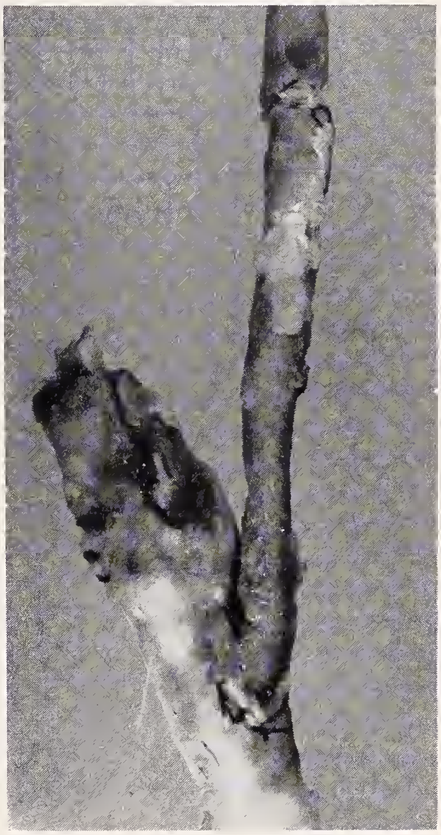

Fig. 5. A side-to-end human homograft which was found to be occluded.

In patients having partial occlusions of the carotid bifurcation, with symptoms of cerebrovascular insufficiency, excision of the atheromatous plaque has been attempted in seven instances. In order to establish the fact that the cerebral circulation is insufficient because of the presence of this stenosis, compression of the opposite carotid should result in dizziness or syncope in ten to fifteen seconds. It is also important to compress the affected vessel to see whether or not complete occlusion will result in increasing the symptoms or signs. This will help evaluate the extent of the insufficiency and may demonstrate any untoward effects from the complete temporary occlusion of the stenosed vessel during the operation, or postoperatively by thrombosis. Compression of the carotid artery on the side of the stenosis in the seven cases above alluded to, did not result in any untoward effects. If compression for ten to twelve minutes causes increased weakness of the opposite half of the body from the partially occluded side, then intermittent carotid compression for a period of several days may result in an improvement in this state. If not, such a patient should be operated under hypothermic conditions to minimize the effects of the ischemia of the brain. The acutely paralyzed patient who shows an occluded carotid in an emergency angiographic study should probably be operated under hypothermic conditions.

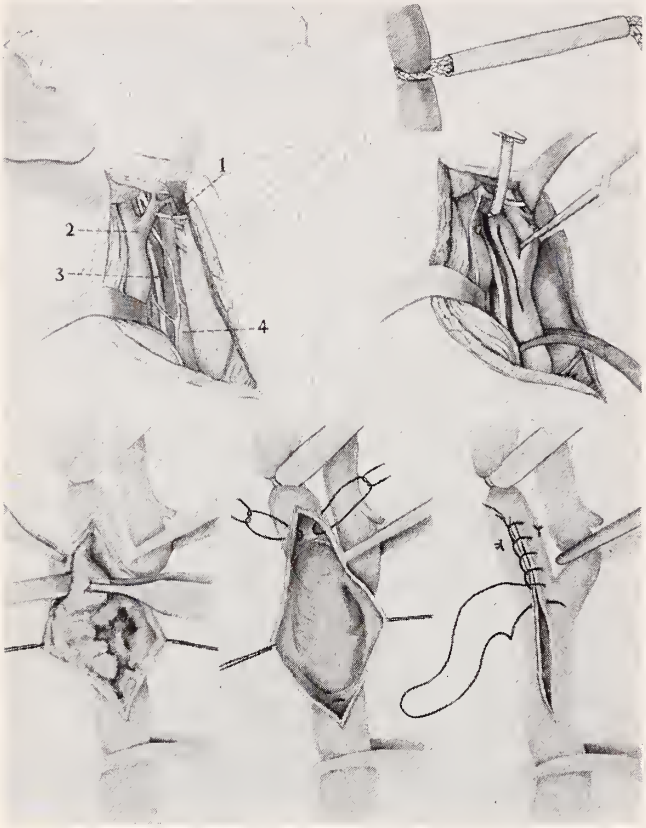

Fig. 6. Technique of intimectomy is illustrated.

Under intratracheal anesthesia, the carotid bulb area and the internal and external carotid arteries are exposed. The deep facial vein emptying into the internal jugular is doubly ligated and cut in order to give as much exposure superiorly under the lower jaw as possible. The twelfth cranial nerve can be retracted superiorly, giving a little more room in exposing the internal carotid artery in this area. In some instances it is conceivable that the twelfth cranial nerve may have to be sectioned, although this has not been necessary in our cases. If section is done, then a suture of the nerve afterward, before closure of the wound is of course indicated. Branches of the ansa hypoglossi usually are sacrificed. The adventitia of the carotid bifurcation and the internal carotid artery is not disturbed in order that this layer be available for suture closure of the incision in the wall after excision of the atheromatous intimal layer. The vagus nerve is identified and exposed.

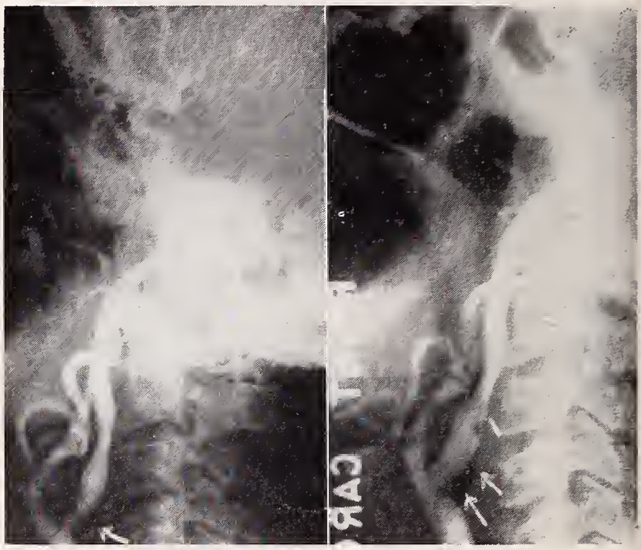

Fig. 7. *(Left)* Angiogram before intimectomy showing a marked stenosis of the internal carotid artery at the bifurcation. *(Right)* Angiogram eleven weeks after intimectomy showing patent internal carotid artery which now does not show the stenosis.

Suitable arterial clamps are used to occlude the proximal and the distal portion of the vessels. In order to occlude the distal portion of the internal carotid artery, a Number-4 braided silk or a tape may be placed around the artery, then passed through a section of polyethylene tubing to form a loop, then tightened about the vessel. This provides more room for working. For occlusion of the common and external carotid arteries, we use long handled bent clamps so that they lie outside the field of operation. If a hypersensitive carotid sinus reflex is present, xylocaine solution is infiltrated about the bulb before any manipulations are begun. Also, 1/50th to 1/100th grain of atropine sulfate is used intramuscularly or intravenously to protect such a patient. We have not used pumping of blood from the common carotid to the internal carotid (distal to the clamps) due to the restricted flow which existed pre-operatively.

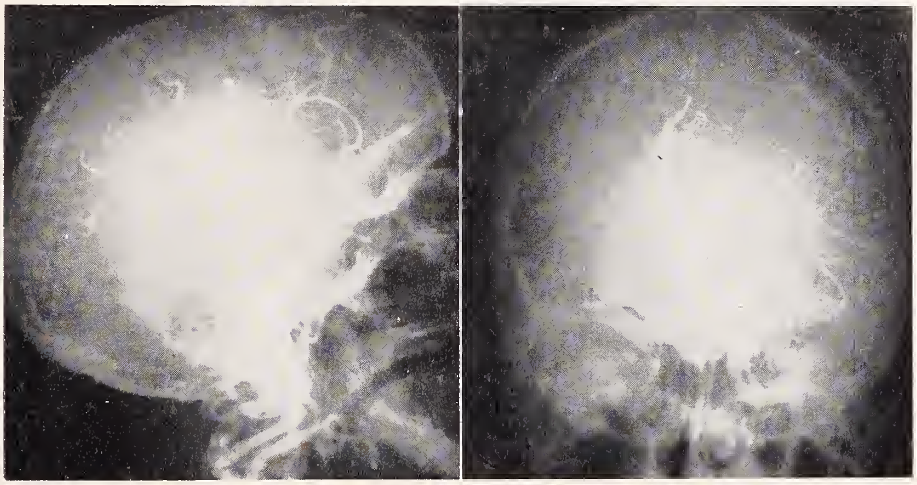

Fig. 8. Carotid angiogram showing left parietal intracerebral mass which proved to be a large intracerebral hematoma. This patient's right-sided paralysis and aphasia completely disappeared within two months after removal of the clot.

Through a linear incision the area of stenosis is exposed; a plane of cleavage is readily identified and the stenotic material internal to the lamina of the internal elastic membrane is removed. The area is flushed with saline and heparin solution and horizontal mattress stitches of 04 waxed arterial suture are used for closing the incision. Air and other matter are permitted to escape out of the vessel before completing closure. In instances where the atheromatous intima is not restricted to the area of the excision, it may be important to suture the intima to the adventitia with several sutures to keep the intimal lining from being dissected by the flow of blood. A strip of gelfoam is held over the sutured vessel using temporary pressure with cottonoid. In a few minutes the incision becomes sealed.

If this method of management is impossible because of the extreme friability of the artery wall, then a homologous graft is suggested.

Before closing, a few words should be said concerning surgical hematomas causing the stroke syndrome. Massive intracerebral hematomas may be readily diagnosed by angiographic studies. Such an example is shown in Figure 8. In this patient a 40-gram clot was removed with excellent clinical results, from the left parietal lobe immediately lateral to the ventricle. Although in many instances there may be extensive deficits in neural function, in a few, spectacular results may be obtained.

Conclusions

1. Evacuation of a massive intracerebral hematoma may be worthwhile.
2. In complete and chronic occlusions of the internal carotid artery, excision of a portion of the vessel and cervical sympathectomy may be valuable.
3. In partial occlusions at the carotid bifurcation, intimectomy in many cases can re-establish the blood flow. Extreme caution should be exercised in evaluating the results.
4. The mechanical removal of occlusive atheromatous formations to maintain the cerebral circulation and to delay cerebrovascular failure must be considered until atheromatous disease of vessels can be prevented or ameliorated by other means.

Surgical Treatment of Aneurysms and Occlusive Disease of the Abdominal Aorta

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SIR ASTLEY COOPER introduced the surgical treatment of abdominal aneurysms when he first performed ligation of the abdominal aorta for an aneurysm in 1817, but little progress was made for many years thereafter. Although advances of a practical nature have been made only relatively recently, the underlying principles of these advances were considered and were rather well developed by experimental workers and applied by a few surgeons near the turn of the century. The notion that segments of arteries could be excised and continuity restored by end-to-end anastomosis, or by the use of arterial grafts, was conceived more than fifty years ago.

Aneurysms of the Abdominal Aorta

The vast majority of abdominal aortic aneurysms occur secondary to ateriosclerotic changes in the wall of the aorta, which cause it to be weakened and permit gradual stretching of the wall and dilatation. In accordance with well-known physical laws, as the wall becomes dilated and gradually stretched, the tension placed on it by the pressure of the blood within becomes increasingly great. Thus, a vicious circle is set in motion that is often finally terminated only by sudden rupture of the aorta and rapid death. This unfavorable prognosis in patients with aortic aneurysms has long been recognized. The studies of Estes[1] indicate that approximately 80 per cent of patients with this disease do not survive for five years, death being due in the majority of patients to rupture of the aneurysm. Somewhat similar findings have been reported by other investigators. Many of those persons who do not die of their disease suffer from severe disability caused by pain in the abdomen and back.

The great majority of abdominal aortic aneurysms arise below the level of the renal arteries. In a recent study reported by my colleagues, Ellis and associates,[2] only 7 per cent of 133 patients operated on at the Mayo Clinic showed involvement of the renal arteries by the aneurysm. It is not possible, however, to determine by physical examination the upper limits of the aneurysm. The aneurysmal sac itself may bulge superiorly from its site of origin and may come to overlap the relatively normal aorta at the level of the origin of the renal vessels. Only by dissection around the superior limit of the aneurysmal sac can the level of its origin be determined. The aneurysms are usually fusiform and may or may not have extensive adhesions and inflammatory reaction about them. Their lower limit may be just above the bifurcation of the aorta, or they may extend to the bifurcation, causing considerable tortuosity of the iliac vessels, or may extend inferiorly to involve the iliac vessels themselves.

As a result, perhaps, of stagnation of blood within the dilated aneurysmal sac in association with calcific and atheromatous changes, a laminated thrombus forms on the inner surface of the aneurysm. The laminated thrombus that lies immediately adjacent to the peripheral wall of the aneurysm gradually undergoes liquefaction necrosis, and at operation one commonly liberates large quantities of gray, oily, liquid material from the aneurysmal wall. This liquefaction necrosis of the thrombotic material supporting and lining the aneurysmal wall is considered an important factor contributing to eventual rupture of the aneurysm.

The clinical manifestations in this disease are characterized by a pulsating mass in the abdomen, accompanied by pain in the upper portion of the abdomen, lower part of the back and the lower extremities. The physical examination is especially important in diagnosis. The mass usually can be

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palpated in the midabdomen at or slightly above the umbilicus, and it often extends more to the left of the midline than to the right. Anteroposterior and lateral roentgenograms of the abdomen usually demonstrate a rim of calcium that is pathognomonic of abdominal aortic aneurysm. Although aortography does have a role in the diagnosis of abdominal aortic aneurysms, it is unnecessary in most cases. Only in extremely doubtful situations, as with obese patients, is such a study indicated.

All patients who have aneurysms of the abdominal aorta should be seriously considered as candidates for operation. Patients who have severe coronary arterial disease may be advised not to undergo operation. Other serious diseases carrying a worse prognosis than that of an abdominal aortic aneurysm may be present and contraindicate surgical intervention. Evidence of leakage from the aneurysm constitutes an urgent indication for operation, although the risk of surgical treatment under these circumstances may be considerably increased. Age, alone, is no contraindication to operation but is of importance, in that persons of the older age groups who have small asymptomatic aneurysms should be less favorable candidates for operation.

The technique of the operation itself is largely standardized. A long midline abdominal incision is made, extending from the symphysis pubis below up to and along side the xiphoid process above, skirting the umbilicus to its left. By eviscerating the small intestine and placing it in a Lahey type of bag on the lateral abdominal wall, excellent exposure of the operative field is obtained. The transverse colon is reflected superiorly, and the terminal portion of the duodenum and the proximal portion of the jejunum in the region of the ligament of Treitz are mobilized to the right. An incision is made in the posterior peritoneum, thus exposing the aneurysm itself. The proximal segment of aorta is dissected free just below the level of the left renal vein, and a tape is placed about the aorta at this point. Similar tapes are placed about each common iliac artery below and the dissection is continued along the lateral margins of the abdominal aneurysm, with ligation of the inferior mesenteric artery. The mobilization of the aneurysm from the underlying spinal column and the lumbar vessels is accomplished only after the aorta is divided above and reflected inferiorly. Special care must be exercised to protect the iliac veins as they pass beneath the bifurcation of the aorta. Following removal of the aneurysm, reconstruction of the aorta must be accomplished. This may be done by utilizing a homograft of abdominal aorta, including the bifurcation; however, particularly when the proximal aorta is dilated, it may be advantageous to use a homograft of thoracic aorta, creating an end-to-end anastomosis above and an end-to-end anastomosis to one of the iliac vessels below. The other iliac vessel is anastomosed end to side.

A transition perhaps is occurring from the widespread use of homografts to the more common use of prosthetic grafts. The experience at the clinic to date with the Edwards-Tapp prosthesis has been most encouraging, and it or a similar prosthesis probably will find wider and wider application.

The afore-mentioned report of the results of surgical treatment of abdominal aortic aneurysms at the clinic included data on 133 patients undergoing operation; at exploration, the condition in ten of these was considered to be inoperable because of involvement of the renal arteries. A mortality rate of 13 per cent was experienced in 103 operations in which an aortic homograft was used. A mortality rate of 35 per cent occurred among patients undergoing emergency operation because of a ruptured aortic aneurysm. Six patients for whom homografts were employed died of postoperative hemorrhage. In one of these patients, the source of bleeding could not be found. Hemorrhage occurred from the suture line in four patients, and there was frank failure of the graft, with rupture through its midportion on the eighth postoperative day, in one instance. Similar experience in other reported series of cases suggests that perhaps tissue incompatibility may be an etiologic factor in the failure of grafts in these patients. As regards the long-term results of the treatment of abdominal aortic aneurysms, Dr. Lincoln Sheranian, a fellow of the Mayo Foundation, recently reviewed these statistics and has shown that the long-term outcome is exceedingly favorable.

Chronic Occlusive Disease of the Abdominal Aorta

Chronic occlusive disease of the terminal portion of the aorta or the iliac arteries is closely akin to abdominal aortic aneurysms as regards the etiologic factors. However, the formation of thrombi occurs initially in occlusive disease. The vessels become narrowed as the result of progressive atherosclerosis until a critical point is reached and final obliteration by thrombotic material occurs.

Leriche[3] pointed out that clinical experience indicates that one may recognize this syndrome easily by three symptoms, namely: (1) absence of pulsation at the two femoral arteries, (2) noticeable blanching of the lower extremities on elevation and (3) inability of men to perform sexually. Perhaps even more characteristic than this so-called Leriche syndrome is the presence of slowly progressive distress in the calf, thigh, hip or back that is induced by activity and relieved by rest.

Of special emphasis in this disease is the fact that roentgenologic demonstration of the exact extent of the disease is indicated. The upper limit of the thrombosis and occlusion is nearly always sufficiently below the renal arteries to enable surgical relief, but knowledge of the lower limit of the occlusion and also the demonstration of the arterial tree of the leg are of fundamental importance with regard to the operative plan.

The surgical treatment of thrombotic occlusion of the terminal portion of the aorta is directed toward re-establishment of normal blood flow to the lower extremities. It may be unnecessary to resect that portion of the aorta involving the bifurcation, in which case a short segment graft is all that is required. In other instances, the lower anastomoses may be made through the common iliac artery on both sides or through its branches on one side. Becoming more and more widely accepted in the case of segmental arterial occlusion is the bypass type of graft utilizing an end-to-side anastomosis inferiorly, thus allowing both a larger anastomosis to be created and also preventing any diminution of collateral blood supply that may be present.

The results of operation for occlusive disease of the terminal part of the aorta or iliac arteries were reported from the clinic by Welch and coworkers.[4] A mortality rate of 4 per cent was encountered among the seventy-four patients who underwent resection and grafting; 69 per cent of the patients had excellent results, 18 per cent had fair results and only 9 per cent showed no improvement. Failures in this series were considered to be due principally to a lack of appreciation early in the series for the necessity of carrying the resection distally until good vessels with good backflow were found, even though this necessitated separate incisions in the groin. Experience has emphasized the necessity of making the anastomosis to the common or superficial femoral artery if the vessels located farther cephalad are not satisfactory. This again serves to illustrate the importance of adequate arteriographic visualization of the vessels in the leg, for an anastomosis to the common iliac artery will fail in a high percentage of cases if the superficial femoral artery on the same side is occluded. Similarly, a graft with an anastomosis to the superficial or common femoral artery may fail if the vessels lower in the leg are occluded.

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(Continued from Page 326)

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Aneurysms of the Thoracic Aorta

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IT IS now well recognized that aneurysms of the aorta, whatever their location, pose a serious threat to life. The development of disabling symptoms and the probability of ultimate rupture are well known. The life expectancy for patients with syphilitic aneurysms of the thoracic aorta has been estimated to be six to nine months.[1] Similar statistics for thoracic aneurysms of other varieties are not available but it is likely that thoracic aneurysms of arteriosclerotic, traumatic or congenital origin also carry a poor prognosis.

Although surgical treatment of a thoracic aortic aneurysm was undertaken more than fifty years ago,[2] this approach has been revived only recently.[3] Probably the most significant stimulus to the development of aortic resectional surgery has been the introduction of the clinical use of aortic homografts by Gross and associates[4] and, more recently, the development of suitable prosthetic materials.[5] Experience with the treatment of thoracic aortic aneurysms at the Mayo Clinic dates back to January, 1954, and forms the basis for this report.

Types of Aneurysms

Twenty-four patients with this disease have been treated. Their ages ranged from five to seventy-three years. Almost two thirds of them were in the sixth, seventh and eighth decades of life.

A wide variety of aneurysms are represented in this relatively small group of patients (Table I). Unlike most reported series, in which syphilitic aneurysms predominate, arteriosclerotic aneurysms were the most common. As the over-all age of the general population increases, it appears likely that an even greater proportion of arteriosclerotic aneurysms will be encountered. They are usually fusiform in shape and more often involve the distal part of the aortic arch and the descending aorta than they do the proximal portions of the aorta. Ten patients had aneurysms of this type (Fig. 1 and 2).

There were five syphilitic aneurysms in this group of patients. Classically, aneurysms of this type occur in the ascending aorta and the aortic arch. Although they are often saccular and have a narrow mouth suitable for lateral excision and aortorrhaphy, these characteristics were not present in any of these five patients.

TABLE I. TYPE OF THORACIC AORTIC ANEURYSMS: 24 Surgical Cases

Type	Cases
Arteriosclerotic	10
Syphilitic	5
Traumatic	4
Congenital	2
Mycotic	2
Dissecting	1
Total	24

Traumatic aneurysms were noted in four patients. Aneurysms of this type are located most commonly in the descending thoracic aorta just distal to the origin of the left subclavian artery at the site of the ligamentum arteriosum. Another common site is the ascending aorta just distal to the aortic valve. Both these sites are relatively fixed points and are placed under stress during rapid deceleration. As the number of automobile and airplane accidents increase, such aneurysms will be seen and recognized with increasing frequency. The surgical results should prove to be good in patients with traumatic aneurysms. They are usually younger than patients with arteriosclerotic or syphilitic aneurysms and the adjacent aortic wall is usually of good quality.

Two patients had congenital aneurysms. These aneurysms are rare. They may be of the aortic-sinus type or they may arise in the region of the ligamentum arteriosum. Both patients had aneurysms of the latter variety.

Two patients had mycotic aneurysms; one occurred distal to a zone of coarctation and one

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occurred at the suture line following resection of a coarctation of the aorta and end-to-end anastomosis performed elsewhere (Fig. 3 and 4).

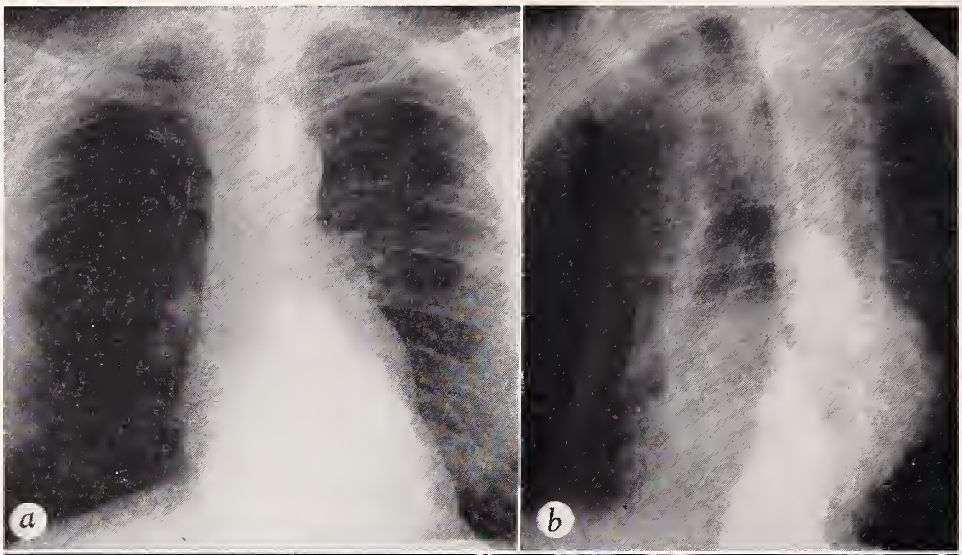

Fig. 1. Arteriosclerotic aneurysm of the descending thoracic aorta in a fifty-seven-year-old man. (a) Posteroanterior roentgenogram. (b) Left anterior oblique projection.

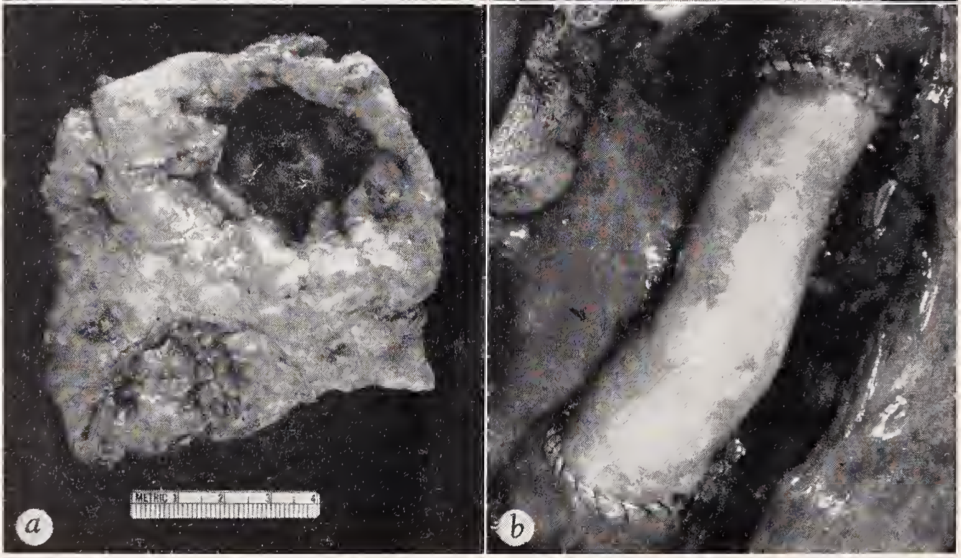

Fig. 2. (a) Opened aneurysmal sac after surgical excision in case illustrated in Fig. 1. (b) Homograft in place in same case.

The final patient had a dissecting aneurysm of the proximal portion of the descending aorta.

Operative Experience

Resectional operations for aneurysms located in the thoracic aorta pose certain problems not common to similar procedures carried out on the abdominal aorta. It is not feasible to place clamps across the thoracic aorta for long periods because of the danger of ischemic damage to the spinal cord. The safe period of aortic occlusion has been estimated to be fifteen to thirty minutes. The great variability in blood supply to the cord may make even briefer periods of occlusion risky. Therefore, various techniques have been employed to avoid this complication. Lateral excision and aortorrhaphy may be employed. This technique may be particularly suitable in the region of the ascending arch, since even brief periods of interruption of the circulation to the brain are not well tolerated. Excision of the aneurysm with insertion of a graft is, however, a preferable procedure, because aneurysmal recurrence at the suture line of the lateral aortorrhaphy was noted in the one patient treated in this manner at the clinic.

The use of temporary shunts to bypass the operative region allows continued circulation to the body during resection. This method was used in one case and was found to have certain disadvantages. The operative procedure is necessarily prolonged and suture closure of the sites of connection of the temporary shunt may be difficult and dangerous, particularly if the entire aorta is diseased, as is often the case. Hemorrhage from one of these sites resulted in the death of one patient.

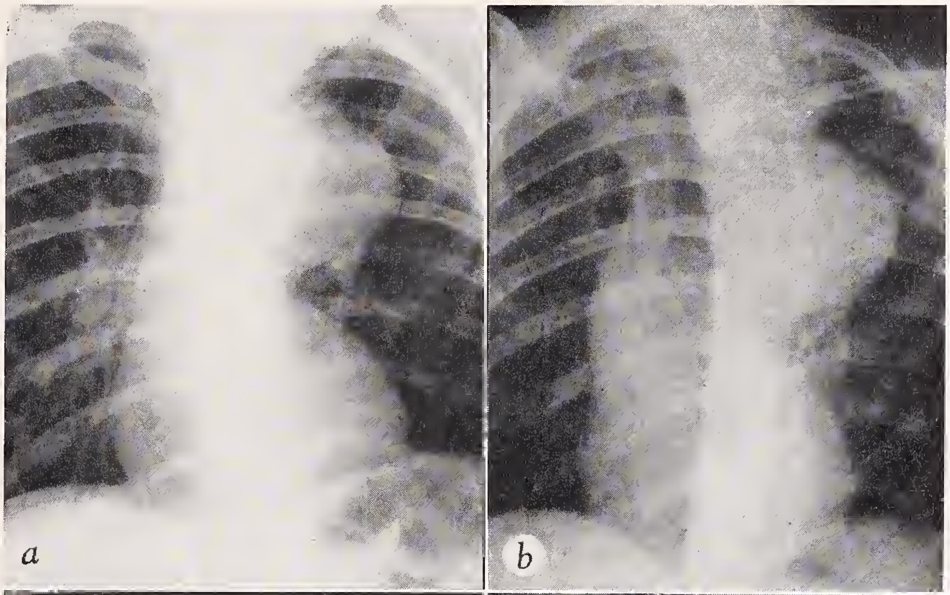

Fig. 3. Mycotic aneurysm in a six-year-old boy after resection of coarctation and end-to-end anastomosis. (a) Posteroanterior roentgenogram. (b) Left anterior oblique projection.

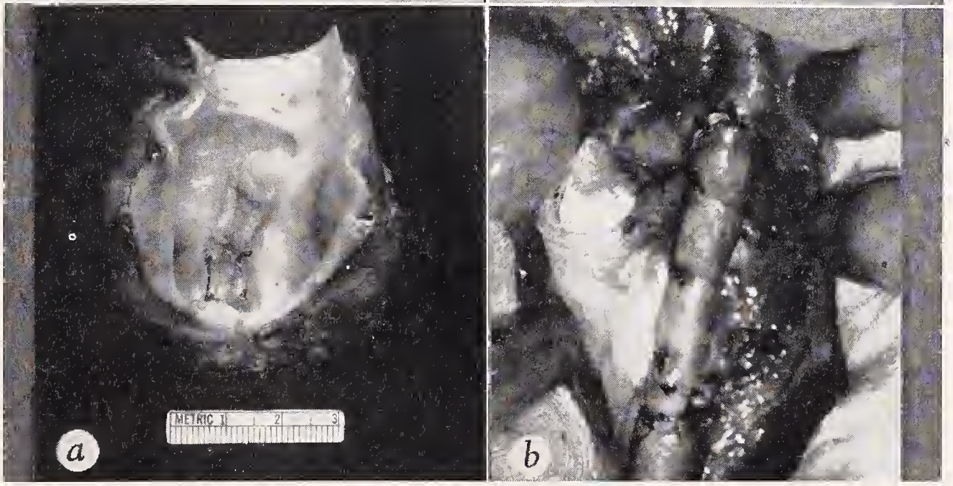

Fig. 4. (a) Opened aneurysmal sac in case pictured in Fig. 3. (b) Homograft in place in same case. (Figures 3a and 4 reproduced with permission of the publisher and authors from Martin, W. J., Kirklin, J. W. and DuShane, J. W.: Aortic aneurysm and aneurysmal endarteritis after resection for coarctation: Report of a case treated by resection and grafting. J.A.M.A., 160:871-874 [Mar. 10] 1956.)

Hypothermia was employed in most of the remaining patients undergoing resection and grafting. By reduction of the arterial oxygen requirement of the central nervous system, a safe period of aortic occlusion up to one hour is permitted. Hypothermia is probably not necessary for lesions located below the eighth or ninth thoracic vertebra. A temperature of 32° C. is ideal and avoids some of the complications of hypothermia that occur at a lower temperature.

Although hypothermia has been a great aid in operative procedures on the thoracic aorta, it has not solved all the problems. The increased work load imposed on the heart by high aortic occlusion can be of serious consequence. In an effort to overcome this, Cooley and associates[6] suggested the use of controlled extracorporeal circulation. By pumping blood from the left atrium to the lower part of the aorta via the femoral artery, blood flow to the lower portion of the body is maintained during aortic excision and grafting.

Resection of the lesion was done on eighteen of these twenty-four patients with thoracic aortic aneurysms. The main cause of inoperability was the location of the aneurysm in either the ascending aorta or its proximal arch. A few instances of successful resection of aneurysms in this location with the aid of extracorporeal circulation have been reported.[7,8] The risks accompanying surgical intervention in this region, however, are obviously greater than when the more distal portions of the aorta are involved.

Grafts were used in seventeen of the eighteen patients on whom resection was done. As previously mentioned, lateral excision with aortorrhaphy was employed on one occasion. There were six deaths in this group of eighteen patients in the first weeks after operation. Hemorrhage, complications of hypothermia or both accounted for most of the deaths. In only one patient were neurologic signs and symptoms recognized in the postoperative period. This patient, operated on under hypothermia, underwent a prolonged period of aortic cross-clamping. Weakness of the lower legs developed, from which he has recovered almost completely. Three other patients died later, all as a result of hemorrhage: one had a ruptured recurrent thoracic aneurysm following lateral excision and aortorrhaphy and two bled because of complications related to the grafting procedure.

Homografts were used predominantly, fourteen patients being treated in such a fashion. An ivalon graft was employed in two patients and a crimped nylon tube in one. The choice of grafting material is controversial. It has been shown experimentally that there is a greater tendency for homografts to undergo degenerative changes when inserted into the thoracic aorta than when placed in the abdominal aorta.[9] For this reason, prosthetic grafts may have certain advantages, although final proof is lacking. However, a greater tendency exists for bleeding to occur through the interstices of a prosthetic graft inserted into the thoracic aorta with the patient under hypothermia than when similar materials are used in the abdominal aorta under normothermic conditions.

Solutions unquestionably will be presented for many of the problems now connected with thoracic aortic surgery as understanding of these problems increases. In the foreseeable future, however, one will have to accept a higher mortality rate for resection of thoracic aortic aneurysms than for resection of aneurysms located in the abdominal aorta. Physicians must ask themselves if operations attended by a risk of this magnitude are justified. In view of the known poor prognosis of untreated aneurysms, surgeons are obligated, I believe, to undertake heroic measures for their correction.

Summary

Aneurysms of the thoracic aorta are a serious threat to health and life, and their surgical removal should be considered seriously.

A series of twenty-four operations for thoracic aortic aneurysms are reported. Arteriosclerotic aneurysms were the most commonly encountered. Resection was performed on eighteen patients, and grafts were employed in seventeen of these, homografts being used in fourteen patients, ivalon grafts in two and a nylon prosthesis in one.

Hypothermia was used as a means of avoiding ischemia of the spinal cord during cross-clamping of the aorta in most instances.

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Effects of Irradiation of the Fetus

Ten-year Follow-up of Pelvimetry during Pregnancy

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AS WE advance into "the atomic age," we are becoming increasingly aware of the possible harmful effects of radiation energy on all living tissue. Especially sensitive to these effects are fetal tissue and the gonads. The public is besieged with implications of the dire results of undue exposure to ionizing radiation, the publicity being based chiefly on the measurable destruction at Nagasaki and Hiroshima where careful and accurate studies[1] have clearly documented in detail the harmful effects of atomic radiation on the population of those two cities.

The literature contains evidence of the unfavorable results of exposure to ionizing radiation[2] in the production of cancer of the thyroid gland or leukemia in patients who, as children, received x-ray treatment of an enlarged thymus gland. The increased incidence of leukemia in roentgenologists[3] as compared with the incidence in physicians not employing roentgen rays has been cited. There is evidence of an increased incidence of leukemia in British and Dutch patients with ankylosing spondylitis[4] treated repeatedly by irradiation. Stewart and co-workers[5] from England, in a recently reported study (in which an unusually large number of children were found to have leukemia or malignant disease following exposure to x-ray radiation during fetal life), have raised the question of the hazard of subjecting any pregnant mother to this exposure. At the 1957 meeting of the British Medical Association. Warrick[6] pointed out three kinds of potential danger to the fetus receiving roentgen radiation in the prenatal period: (1) occurrence of congenital deformities as a result of radiation injury during the first trimester, (2) damage to the gonads of mother and fetus resulting in gene mutations that might appear only in future generations and (3) the production of leukemia or malignant disease during the first 10 years of life from repeated small doses of irradiation. All of these disadvantages must be weighed against the value of information gained by obstetric radiology.

A study by the National Research Council[7] in 1956 of the biologic effects of atomic radiation suggested that the total man-made radiation to the gonads in an entire reproductive lifetime (birth to 30 years of age) should be kept less than 10 r to avoid harmful mutations of the genes. We are now receiving 3 to 4 r, on the average, from medical x-ray exposure, which is the same dose as unavoidable background radiation in a lifetime. The fall-out from the testing of atomic weapons is minimal, probably less than 0.5 r. The effect on all of us of the output of atomic energy plants is a matter of conjecture. A total dose of 200 r in a lifetime may be harmless if distributed over the years with a weekly dose of less than 0.3 r, according to Witts.[8] The average pelvimetric x-ray examination delivers a dose of 2.5 r to the fetus, according to Rabinowitch.[9] This is a dose in the range that may be harmful to the fetus and maternal gonads.

The previously mentioned report of Stewart and colleagues,[5] made just a year ago, has caused much concern to all confronted with this problem. In a survey of 1,500 children dying of leukemia or malignant disease before the age of ten years in the period 1953 to 1955, they found in the first 500 cases eighty-five children whose mothers had had x-ray exposure of the abdomen during the antenatal lives of these children, as compared to 45 children in a control group. This evidence suggested to Stewart and co-workers that children who receive x-ray irradiation before birth are

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Read at the meeting of the Northwestern Pediatric Society, Bayport, Minnesota, September 27, 1957.

Dr. Mills is in the Section of Pediatrics, Drs. Bruwer and Davis are from the Section of Roentgenology, Dr. Banner is in the Section of Obstetrics and Gynecology and Mr. Gage is from the Section of Biometry and Medical Statistics, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

more prone to have leukemia or malignant disease subsequently than are those who have not been exposed to x-rays *in utero*. So challenging was this report that we were prompted to review our own experience in this connection, since it has been the practice of the Section of Obstetrics of the Mayo Clinic to use x-ray pelvimetry in many primigravidas to corroborate pelvic measurements. Pelvimetric x-ray examination may also be employed in multiparous patients having delivered elsewhere, who have delivered infants of less than average weight, or who give histories of dystocia possibly due to cephalopelvic disproportion.

The review of our own experience concerns 226 of 1,258 patients delivered in the hospital in 1946. These 226 patients had received x-ray pelvimetry during their pregnancies. This procedure had been carried out usually in the last trimester both to include those mothers expected to deliver at term and also to avoid the greater danger of inducing congenital malformations that may be induced early in gestation. The dose of radiation delivered at the depth of the fetus by pelvimetry in 1946 was calculated as 3.5 r. As this is more than the dose previously mentioned as harmful to the fetus we would expect any damage to the fetus to be evident in a study of the subsequent course of these children. Of the 226 children represented by the study, we had a complete 10-year record on 155, in none of whom was there any evidence of malignant disease or leukemia. The abnormalities and deaths in the 155 are summarized in the table. Also in an additional thirty-five, followed for varying periods of less than ten years, we could find none with evidence of leukemia or malignant disease. The remaining thirty-six patients could not be traced following birth.

Although this sample is small, the ten-year follow-up of 155 of the total 226 children (almost 70 per cent) who received x-rays antenatally gives no evidence to support the suspicion of an increased incidence of leukemia or malignant disease in such patients as suggested by Stewart and coworkers. Had our patients followed the pattern suggested by the British authors, we might have found some evidence in a ten-year period among the 190 traced.

To obtain some perspective of the problem it might be well to state that the death rate from cancer including leukemia for children less than fifteen years of age in the total registration area of the United States in the year 1952 was approximately ten per 100,000 population of that age period. Stated differently, there were slightly more than 3,600 deaths in children less than fifteen years of age in the entire United States from all kinds of malignant neoplasms and leukemia in the year 1952. Though the incidence of these conditions is relatively large in childhood, the actual number of patients who die of them is small considering the size of the population in which they occur. In view of this low incidence, it is not unexpected that a series such as ours, which comprised only a small number of patients, contained no cases of malignant diseases or leukemia.

TABLE I. ABNORMALITIES IN 155 CHILDREN TRACED FOR TEN YEARS AFTER ANTENATAL IRRADIATION
(Pelvimetry Done in 1946)

Abnormality	Patients	
Stillborn	2	
Birth injuries—died	2	
Prematurity—died	1	
Congenital deformities	3	
Coxa vara		1
Deformity of cervical spine		1
Microcephaly		1
Congenital deafness	1	
Congenital cataracts	1	

Rabinowitch[9] refuted the implication of Stewart and colleagues that there is a positive correlation between roentgen irradiation given antenatally and the occurrence of leukemia and malignant disease in the children subjected to such irradiation. He checked the records of all children dying of leukemia in his hospital area from 1940 to 1955. There had been more than 4,000 roentgen examinations during pregnancy, about a third of which were pelvimetries. There were but six deaths from leukemia in the age group less than six years; none of these children had received antenatal roentgen irradiation. It will require further carefully controlled studies of large numbers of patients to settle this question.

Conclusions

1. The use of x-ray pelvimetry in the third trimester of pregnancy appears to have shown no tendency to cause malignant disease or leukemia as a result of radiation injury to the fetus.

2. The advantages of x-ray pelvimetry to the obstetrician would seem to justify the very limited risk of potential injury to the fetus that is entailed in the use of the procedure.

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Discussion

Marvin M. D. Williams, Ph.D. (Rochester, Minn.): The general subject of this paper is not only of scientific interest at the present time but is included in national and international political issues and is being publicly debated, discussed and argued. Scientific knowledge regarding the biologic effects of radiation is scanty, particularly regarding the effects on humans, because of the difficulty in obtaining accurate and controlled data. Soon after their discovery a little more than 50 years ago, roentgen rays were found to produce some injurious biologic effects. Since that time, the philosophy relative to the problems of protection from radiation has undergone continuous evolution as knowledge regarding radiation effects and as the number of people being exposed to radiation have increased.

At first, the dangers were thought of only in relation to persons actively using x-rays and were often considered to be a necessary occupational hazard. When it was realized that others working near the x-ray machines also were being exposed, the possibility of providing some protection began to be considered. As the use of x-rays expanded and greater numbers of persons were likely to be exposed to radiations, including many who understood little or nothing about either the radiations or their dangers, it was recognized that protection must be provided and that standards of protection should be decided on. Definite efforts to determine and specify protective procedures and permissible exposures were undertaken in the 1920's. In 1928, the National Committee on Radiation Protection was organized under the sponsorship of the National Bureau of Standards, and this committee has continuously studied the problems of radiation protection and has made recommendations.

The present recommended permissible dose is less than the one specified in 1928 by a factor of between 10 and 20. The larger 1928 dose never has been shown to have produced a detectable effect in a person, but knowledge gained since then indicated that the factor of safety may have been small; therefore, it appeared advisable to reduce the recommended permissible dose. The last reduction in this dose, made about a year ago, also took into consideration the increasing number of people receiving occupational exposure, an anticipated increase in the exposure of the entire population (from such sources as shoe-fitting x-ray machines, mass thoracic x-ray survey programs, and the by-products of nuclear reactions), and the possible genetic effects that might be produced.

The problem of chief concern now is not so much the effects of radiation on the individual himself as it is the possible genetic effects that may appear perhaps 50 generations hence. The probability of genetic effects being produced depends more on the total dose received by the entire population (up to the end of the reproductive period) than it does on the dose received by any one person. The present permissible dose is believed to be less than the minimum necessary to produce detectable injury in a person, but the magnitude of the genetic effects that may be produced by even much smaller doses is still not known.

Although values for the permissible dose have been suggested by the National Committee on Radiation Protection, it must be kept in mind that there is no implication that such a dose will produce no undesirable effects, and also that it is undesirable for any person to receive such a dose unnecessarily. It is believed that no provable detectable injury will be produced in a person if his exposure is consistently less than, or even if it somewhat exceeds, the permissible dose. However, such a dose could be responsible for genetic effects in future generations and might perhaps tip the scales so that the person could be more susceptible to some disease, possibly thereby contributing to a shorter life span. More likelihood exists of damage to children than to adults from small exposures; during the first 3 months of pregnancy, the fetus is more susceptible to radiation injury than it is thereafter.

Radiation can be extremely useful as a diagnostic tool. The problem is to reach a satisfactory and reasonable compromise between the possible injuries that may become evident in the future and the probable immediate benefits to the person concerned (some additional factors enter into the application of radiation to treatment that are not relevant to this discussion). It must be remembered also that failure to use roentgenologic diagnostic procedures may cause greater harm than would be caused by the radiation.

The problems of determining what are the acceptable uses of radiation, what are permissible exposures to the entire population and what is a permissible exposure to an individual are all interrelated and also are related to many moral questions and national and international issues. Compromises are necessary, and the problem must be decided now on the basis of the present somewhat scanty knowledge. Changes will be necessary in the future, as they have been in the past, as knowledge increases and as other factors change.

The information that this study has contributed to the effects of radiation, when added to the other information already known and that to be gained in the future, will aid in the satisfactory solution of the complex problems of radiation protection. The members of this audience have an opportunity and a responsibility to continue to accumulate such data.

The Pancreas and Diabetes Mellitus in Man and Animals

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THE PANCREAS has long held a unique place of interest relative to clinical and laboratory studies of diabetes mellitus, since it is the only known source of the antidiabetic hormone, insulin. Its position in this regard is still a central one in spite of the recognized importance of other body factors in the initiation and course of diabetes. In this paper, attention will be limited to those studies on the pancreas and insulin being made by one of the fifteen research groups housed in the Charles H. Best Institution at the University of Toronto, the team of which I am a member. When I speak of "we" in what follows, I shall be referring collectively to members of this team.

We have been occupied in recent years with two distinct types of research on the pancreas and diabetes. On the one hand, this has included studies on the effects of insulin on the rates of transfer of metabolic factors into and out of various compartments of the living body. On the other hand, factors and conditions influencing the amount of insulin extractable by a standardized method from animal and human pancreas have been under study.

It is possible to represent a living system as an extra-cellular transfer medium in which are located groups of cells or formed elements within specific tissues between which transfer of metabolites can occur only by way of the extracellular fluid. Sheppard and Householder[15] have called such a centrally-exchanging arrangement a "mamillary" system and have developed a mathematical basis for measuring rates of transfer between compartments in such a closed system in dynamic equilibrium. We have extended their methods to measure simultaneously the rates of transfer of metabolic elements into and out of all compartments of an organism by means of isotopic tracers in the absence of dynamic equilibrium.[16] This theory has been used to measure the rates of transfer of phosphorus into and out of sixteen organ compartments in the normal rat without assuming that dynamic equilibrium exists or that rapid intermixing of phosphorus occurs in the organ compartments.[10] An example of such a mammillary system is shown in Figure 1.

The validity of this theory has been tested in a number of ways. In the first place, model hydrodynamic systems have been set up in which rates of water transfer and amounts of water in the compartments of a system were measured both directly and by means of the tracer method. The two sets of rate measurements were then compared and their magnitudes were found to agree within the limits of experimental error.[11] Secondly, it has been possible to calculate some transfer rates in mammillary systems in two ways, and these calculated rates have been found to cross-check quantitatively, both in hydrodynamic systems[14] and in the measurement of transfer rates of phosphorus in the normal rat.[10] The measured amounts of extracellular phophorus in the normal rat also have been calculated in two ways, and these calculated values have been found to cross-check closely.[10] From these measurements, the amount of intracompartmental phophorus in each of the sixteen tissues studied has been calculated. It appears likely that the intracompartmental phosphorus calculated in this way represents the intracellular phosphorus in cellular compartments, but this remains to be proven.

Measurements of transfer rates of phosphorus in alloxan-diabetic rats with and without insulin therapy have just been completed,[8] and here again the results cross-check satisfactorily. These experimental results indicate that insulin acts in the alloxan-diabetic rat to increase the rate of transfer of phosphorus from the plasma compartment into all tissues except the gonads. The increase is greatest in the cardiac and skeletal

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muscles and, percentagewise, is appreciably less in the liver.

The same tracer methods for analysis have been used to seek a direct answer to the question ,"How does insulin lower the blood sugar in the fasting, insulin-deprived, depancreatized dog?" It is recognized that two possible modes of action exist. Insulin may act to increase the removal of sugar from the blood into the tissues or it may act to decrease the release of sugar into the blood by means of gluconeogenesis. Both an abrupt and large increase in the rate of removal of glucose from the blood plasma and also a slow but cumulative decrease in the rate of gluconeogenesis followed the intravenous injection of insulin into the depancreatized dog deprived of insulin for sixty-six hours.[7]

These findings are of interest when compared with those of Weinhouse's group,[4] who made a similar study of the effect of insulin introduced into the blood plasma of normal dogs on the supply and removal of blood glucose. Their findings indicate clearly that the rate of influx of glucose to the blood of the normal fasting dog falls to zero when insulin is administered intravenously. A comparison of these findings indicates that a prompt stoppage of glycogenolysis is probably involved in the homeostasis of the blood sugar in their normal animals, while the suppression of gluconeogenesis by insulin is a much slower effect in the depancreatized dog.

Dr. Margaret Henderson of our team is at present measuring the rates by which glucose is supplied to and removed from the blood plasma compartment before and after the intravenous injection of another pancreatic extract, glucagon, into the fasting, depancreatized, insulinized dog. Under these conditions, glucagon causes an abrupt increase in both of these rates.

All of our tracer measurements have been made in the absence of dynamic equilibrium, and they represent measurements of the absolute rates and compartment contents involved during the adaptation to a specific stimulus in entire living systems. This kind of measurement is of fundamental interest in physiology but, as yet, is rarely seen in publication. The assumption of dynamic equilibrium made by most workers studying turnover has barred them from this type of observation.

What place do the afore-mentioned studies on the effects of insulin on diabetic rats and dogs have in a department of medical research? There is much solid ground on which the virtue of such studies in such a department can be established. However, rather than to discourse on these, I prefer to turn to the second branch of the work of the section. This requires no such defense since it deals largely with studies on the human pancreas. Standardized procedures for the extraction and assay of insulin from pancreases obtained at autopsy have been followed in this section for several years. The measurements obtained in this way now represent a sizable reference store of data against which the validity of many postulates concerning diabetes in man can be tested. We currently have available measurements on the insulin extractable from the pancreas at autopsy for 100 adult and seventy growing nondiabetic persons as well as for 170 diabetic patients of all ages.

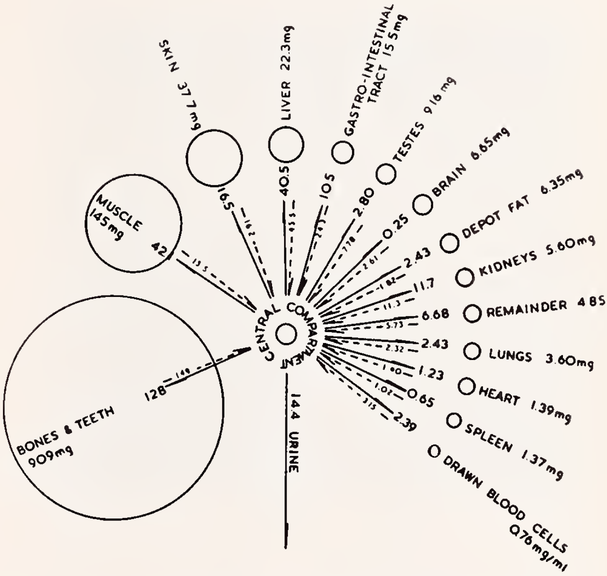

Fig. 1. Rates of transfer of phosphorus (P) in the fasting male Wistar rat with severe alloxen diabetes. All transfer rates are expressed as μg P/Min; amounts of P in each compartment are shown after compartment name.

These measurements clearly indicate that a gross lack of endogenous insulin is characteristic of human beings who acquire diabetes during the period of normal growth (approximately the first twenty years of life). Diabetes diagnosed thereafter differed in that a considerable amount of insulin was present in the pancreas at autopsy in a majority of such persons.[20]

It has long been recognized that the diabetes of most children differs from that in adults in that it is characterized by overheight and emaciation at diagnosis, and it usually is followed by early death in diabetic coma if insulin is not given. Because of these observed differences and because of the differences between the levels of insulin in the diabetic human pancreas during and following the normal growth period in man, the term "growth-onset" diabetes was suggested to resolve the former apparently homogeneous group from the latter heterogeneous group of persons who became diabetic after the state of full stature (maturity) had been achieved.[20]

Fig. 2. Bonny, a purebred Irish Terrier bitch which was diagnosed as diabetic at the age of two years and three months. Her daily ration is 100 gm. of cooked horse meat plus 100 gm. (dry) of K-9 meal. She receives 26 units of protamine-zinc insulin per day.

Not only did the insulin of the pancreas fall to extremely low values within a year of the diagnosis of diabetes, but the number of beta cells also fell to extremely low levels, and granulation in beta cells became practically nonexistent within the same short span of a few months in the growth-onset diabetics of our series.[24] Since positive evidence exists to support the view that the beta cells of the human pancreas are the source of endogenous insulin,[6] growth-onset diabetics appear to be diabetic due to a gross shortage of endogenous insulin.

The spontaneous diabetes of dogs shows partial parallellism to growth-onset diabetes in man.[24] Untreated spontaneous diabetes in the mature dog is associated with severe wasting, cataracts and normal sensitivity to insulin. Six of the eight dogs which had spontaneous diabetes in our studies were bitches. All but one of these were over five years old.

At the request of and with the cooperation of some of the owners, such diabetic dogs have been kept alive and well for varying periods. In every case it was possible to control the diabetes in the laboratory by use of diet and insulin. "Bonny," the youngest surviving diabetic dog on record, lives at home on a measured diet and insulin, and with periodic examinations at the Best Institute. Bonny appears to be healthy and is active (Fig. 2). The diabetes of this dog has been under such treatment and observation for nearly five years.

The insulin extractable from the pancreas at autopsy has been measured in four of the eight spontaneously diabetic dogs studied by Ricketts and associates,[13] as well as in the seven nonsurviving dogs studied in Toronto. The same picture of rapid loss of extractable insulin, of beta cells and of beta cell granulation as was seen for growth-onset diabetics was found to occur in these dogs.[6,13] In these regards, the two types of diabetes appear to be closely comparable.

The alloxan-diabetes of rats also was found to be accompanied by a profound lack of endogenous insulin. Even when the diabetes of such rats had regressed until it was no longer evident, they still had a gross deficit in the amount of insulin stored in the pancreas.[22]

Nothing more than a slight indication of a decline in the average amount of insulin extractable from the pancreas with duration of diabetes for periods of up to twenty-five years has been observed in diabetic patients in whom the diagnosis is made after the normal growth period. This conclusion is based on all such persons on whom studies of insulin in the pancreas have been made; it is recognized that, while the average level of insulin in the pancreas of such persons amounts to almost half of that seen in nondiabetics similarly studied at autopsy, some of these persons have extremely low levels of extractable insulin. Thus, while some maturity-onset diabetic patients appear to have a grossly inadequate reserve of endogenous insulin, many others do not. Studies on the insulin content of blood plasma from diabetic and nondiabetic persons also indicate that lack of endogenous insulin is characteristic of diabetic children, but that frequently the plasma-insulin levels in maturity-onset diabetics are within the range of nondiabetic values.[2]

Is there a form of spontaneous diabetes in animals similar to that in mature man in which gross deficiency of insulin is not seen? Studies have been proceeding with the cooperation of Dr. Jean Mayer and his associates, of Harvard University, on the insulin extractable from the pancreas of the obese-hyperglycemic mouse, with this possibility in mind. In this type of animal, high levels of pancreatic insulin relative to those seen in nonobese control sibs were repeatedly observed. There was no evidence that this form of "diabetes" caused or resulted from a subnormal supply of insulin or that the "diabetes" caused or resulted from reduction in the number of islet cells with long duration of the condition. On the contrary, hyperplasia of islet cells was present.[17] Shortly after such animals received injections of growth hormone, the level of blood sugar was further elevated. At that time, a fall was observed in the insulin extracted from the pancreas of the obese-hyperglycemic mouse, the fall being proportional to the magnitude of the increased elevation of the blood sugar caused by growth hormone.[3]

Several factors in the human pancreas in addition to the extractable insulin have been studied and are under study. The extent of beta cell granulation that could be demonstrated by special histologic stains has been compared with the concentration of insulin extractable from other sections of the same pancreas, and a significant 1:1 correlation between these two factors has been observed for both nondiabetic and diabetic persons.[6] This confirms the similar findings of Bell[1] and represents added evidence that, in human beings, these stainable granules in the beta cells are manifestations of insulin.

During the past two years, considerable interest has attached to the lowering effect on blood sugar in man of orally administered carbutamide and tolbutamide. Extensive studies of this type of response have been made in Europe and on this continent. The findings of Mirsky and coworkers[12] are of special interest in this regard, particularly since they were reached objectively through the application of recognized statistical procedures. These authors measured the decrease of blood sugar during the first six hours after administration of a single oral dose of tolbutamide to 200 living diabetic patients of both sexes and all ages. They concluded that (1) practically all growth-onset diabetic patients except those who are newly diagnosed, are much less responsive to the hypoglycemic action of this drug than are those diagnosed after the age of forty, (2) responses in persons diagnosed as diabetic within the age range of twenty to forty years are intermediate in degree, (3) little relationship exists between effectiveness of response and duration of diabetes in the older patients, and (4) no detectable sex difference occurs in the response to the tolbutamide test.

Striking similarities are noted between these findings and the levels of insulin extractable at autopsy from the diabetic human pancreas. Corresponding to points (1) and (4) of Mirsky's group, it has been reported that the level of insulin extractable from the pancreas in growth-onset diabetic patients falls within months to characteristically extremely low levels in all but newly diagnosed patients,[24] but it is much higher and is insensitive to the duration of diabetes in maturity-onset patients.[20] We recently reported a sex difference with a moderate degree of statistical significance in the insulin extractable from the pancreas in persons diagnosed as diabetic in the twenty to forty-year range.[18,23] The female patients in our series who were diagnosed as diabetic in this age-range had appreciable pancreatic stores of insulin, while the corresponding males had very little. Evidence has been presented[23] indicating that the apparent disagreement with the conclusions of Mirsky's group on this point developed as a result of appreciable differences in the average duration of diabetes in the two series.

Comparisons of the levels of insulin extractable from the diabetic human pancreas with the proportion of diabetic patients responding to oral use of carbutamide have been made on the basis of age at diagnosis and known duration of the diabetes.[18] This comparison, together with the one already described, favors the hypothesis that a significant supply of endogenous insulin is required for carbutamide or tolbutamide to normalize the amount of blood sugar in diabetic man.

We have gathered information concerning the effect of the interval between death and autopsy on the amount of insulin extractable from the human pancreas. Instead of values that declined steadily with the time of aging, an early rise and a subsequent fall were noted in the insulin extractable from samples of human pancreas during aging at 4° C.[6] The same phenomenon has been observed in fresh beef pancreas under more closely controlled conditions[9,19,21] The experiments thus far indicate that the initial rise may result from the synthesis of insulin and its accumulation in the initially fresh pancreas in the absence of circulating blood. The phenomenon appears to be closely related to the similar findings of Grodsky and Tarver[5] observed during the incubation of fetal beef pancreas.

In conclusion, I would like to speak briefly about the team to which I am attached. It is composed mainly of young people, containing approximately equal numbers of men and women. Its members are drawn together from widely distributed parts of the world by common interests in problems of carbohydrate metabolism.

Ideas are precious commodities with which research teams work; unless care is taken to prevent it, the origin of such ideas may become a matter of question after weeks or months. Particular care is taken to avoid such difficulties by maintaining a series of "Idea Books" in which new ideas are duly entered, signed and dated at the time of their origin, regardless of whether such ideas do or do not appear to be important. In this way, a pool of research ideas is open to all members of the team, and ideas beget ideas. There is also an essential spirit of friendly competition among members of the team to register new ideas, and this is good for all concerned.

Not all projects launched by the team have been successful, and I think it is necessary to be frank about it. For example, one student and I worked together for over a year on measurement of the transfer rates of phosphorus in the mouse. The results of this experiment did not cross-check since the tracer phosphorus had been injected in a nonphysiologic form, and the findings had to be shelved.

A very important member of each of the teams currently engaged in medical research at the Institute is Dr. Best. The selection of the various people who make up these teams is in his hands, and the degree to which he is able to keep in touch with the detail of projects that are continuing in the Institute named after him is remarkable. We are indebted to him not only for many valuable suggestions but also for direct participation in some of the experiments and for the "tying together" of our projects and findings with those of other teams and other research centers. He is indeed a leading and contributing member of our team. As one who is diabetic, it is a special privilege for me to be a member of his group in the hunt for a killer that is still at large.

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Case Presentations

Rupture of Vaginal Varicosities Simulating Placenta Previa

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THE LITERATURE carries little on the subject of ruptured vaginal varicosities which resemble placenta previa. The importance of making this differential diagnosis prompted the following presentation.

Case Report

Mrs. K. A., age thirty-two, was seen for the first antenatal visit of her third pregnancy on May 14, 1956. Her last menstrual period had occurred on February 1, 1956 and was normal. The patient complained of amenorrhea, mild nausea, and abdominal "fullness."

General physical examination revealed a well-developed, slight, white woman weighing 114 pounds. Blood pressure was 112/60. A small lymph node was palpable in the right axilla. The breasts were full and slightly tender. The cervix was livid and soft. The uterus was anterior and fourteen weeks' gestational size. There were moderate varicosities of the left leg along the superficial saphenous distribution. Hemoglobin was 13.2 grams/100 ml. Pelvic mensuration and evaluation was done and found adequate for vaginal delivery. Remainder of the examinations were essentially negative. A diagnosis of normal intrauterine pregnancy was made, and the patient was given an E.D.C. on November 8, 1956.

On subsequent prenatal visits no complaints were made except for occasional reference to varicosities in the left leg. These were asymptomatic while using an elastic stocking. There were no complaints or abnormal findings referable to the vulva or vagina.

About 6:00 p.m. on August 29, 1956 (thirty weeks' gestation) the patient sat down on a chair and a pool of blood formed in the chair. She became faint and weak and was put to bed immediately by her husband. An ambulance was dispatched, and the patient arrived at the hospital at 7:15 in the evening. She was admitted to the delivery room and examination revealed moderately heavy vaginal bleeding with large clots formed at the vestibule extending into the vagina. Pelvic examination was deferred until blood was available.

The fetal heart tones were regular and of good quality. There were no uterine contractions and no tenderness was present. The blood pressure was 120/70. One thousand cc. of 5 per cent dextrose in distilled water was started intravenously with a Y-tube transfusion apparatus. Immediate cross match and hemoglobin determinations were ordered. A perineal prep was ordered, during which the bleeding became more severe. The patient was then transferred to surgery where preparation for a Cesarian section had been arranged on the presumption that the diagnosis was most likely a placenta previa, because of the severe painless bleeding in the latter part of pregnancy. At this time (8:30 p.m.) the hemoglobin was reported as 10.4 grams/100 ml., and one pint of blood was attached to the intravenous setup. Bleeding time was reported 1′ 56″ and coagulation time was 15′ 15″. Although the patient appeared pale, the blood pressure maintained itself at 120/60. The vaginal bleeding again was noted to have slowed to a moderate flow.

After some discussion as to whether the routine presurgical vaginal examination should be foregone, it was decided that the fetal and maternal condition would not be jeopardized by this procedure, so this was done. After removal of the clots at the introitus, a vaginal speculum was introduced. It was noted that the upper two-thirds of the vagina was completely free of blood. Further inspection revealed free bleeding from the right postero-lateral vaginal wall just within the hymenal ring. A plexus of varicosities was noted to protrude in this area. The bleeding points were clamped with Kelly hemostats and the varicosities were closed at several points with interrupted sutures tied over the vaginal mucosa. The bleeding was entirely stopped by this procedure, and it was felt that the pregnancy could safely be allowed to progress to a normal vaginal delivery if no further complications were encountered. The total blood loss was estimated to be in excess of 2,000 cubic centimeters.

A second pint of blood was given after surgery was completed. A placentagram was taken on August 30, 1957 and was negative for placenta previa. The hemoglobin on the third postoperative day was 12.0 grams 100 ml. The patient made an uneventful recovery and was discharged on the fourth postoperative day (September 2, 1956).

The patient had no further complications. Frequent inspection of the operative site revealed excellent healing with no residual varicosities. Delivery occurred at term (November 6, 1957), without incident. The baby weighed 8 pounds 6 ounces. A midline episiotomy was used. The patient again recovered uneventfully and was discharged on the fourth post-partum day.

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Recurrent Lower Leg Ulcers in Lupus Erythematosus

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IN ORDER to call attention to recurrent infarctive lower leg ulcers as part of the syndrome of lupus erythematosus, the following case report and brief review of the literature are presented.

Case Report

Mrs. G. W., a white housewife, aged forty-five, developed an erythematous eruption on the butterfly area of the nose and cheeks at age nineteen. This eruption was accompanied by swelling and pain in the joints of the hands. A diagnosis of lupus erythematosus, confirmed by cutaneous biopsy, was made by Dr. H. E. Michelson, and treatment with injections of gold salts was instituted. This was followed by temporary disappearance of the eruption and joint symptoms, but there were periodic exacerbations of the skin lesions for the next seven years. At age thirty-five she developed symptoms of numbness in the fingers and toes after exposure to cold. The Raynaud-like symptoms have persisted to date, but have never been very severe. There has been no actual pain or trophic changes in the digits.

At age twenty-eight, Mrs. W. developed the first of a series of leg ulcers. New ulcers have appeared periodically since, at the rate of one or two new lesions each year, so that she has now had a total of over twenty, all near the ankle joints. The ulcers appear rapidly, starting as an area of bluish-red discoloration of previously normal skin. At this stage (and later) there is severe pain in the involved area. There is no swelling, but within a few days the discolored areas become necrotic and an ulcer forms. These ulcers average 2 to 3 cm. in diameter, extend as deeply as the periosteum, and are extremely indolent, usually taking several months to heal.

Prior to the use of corticosteroids in the treatment of these ulcers, a number of different remedies were unsuccessfully tried. These included prolonged bed rest, numerous local applications, enzymatic debridement, a number of different antibiotics, such vasodilating drugs as tolazoline hydrochloride (Priscoline) and dibenzylene, and attempts at skin grafting. In 1945, a right lumbar sympathectomy was performed, following which the ulcers then present on the right ankle promptly healed. Subsequently, however, there have been as many ulcers on the right leg as before, and the patient states that they have been more painful than those on the left leg.

Physical examination shows a slender white woman who appears well except for her leg lesions. There is a slight malar flush, apparently the result of diffuse superficial telangiectasia of that area, but no frank lesions of lupus erythematous. The blood pressure is normal, and the liver and spleen are not palpable. The fingers are somewhat cold, but show no blanching or cyanosis, and no trophic changes. The right leg is somewhat warmer than the left, but good pulsations are palpable in both.

At the present time, an ulcer is present on both ankles, and numerous scars mark the sites of former lesions (Figs. 1 and 2). The ulcers measure aproximately 2 x 3 cm., are sharply punched out, deep, indolent, and filled with necrotic debris.

Laboratory studies have been carried out on several occasions. Hemoglobin, red blood cell count, hematocrit and color index have always been within normal limits. The leukocyte count has varied from 5400 and 6850 cells per cubic mm., with a normal differential count. The urine has been normal except for a reported trace of albumin on one occasion. Serologic tests for syphilis are negative.

Tests for cryoglobulin have given negative results on two occasions. Electrophoretic fractionation of serum proteins gave the following results: total protein 7.6 grams per cent, albumin 3.3 grams per cent, globulin 4.3 grams per cent and gamma globulin 2.1 grams per cent.

Tests for lupus erythematosus (L.E.) cells in the peripheral blood have been done on three occasions, in three different laboratories. The first two tests were negative, but the most recent, carried out by Dr. Dorothy Sundberg, showed L.E. cells.

For the past six weeks this patient has been treated with prednisone, 20 mgm. daily. In spite of the fact that she has not been confined to bed, the ulcers have gradually filled with granulation tissue and have almost completely epithelialized from the edges.

Comment

Recurrent ulcers of the lower legs have not been commonly regarded as part of the syndrome of lupus erythematosus, and have not been mentioned in any review of that disease known to us. However, several indirect references to such an association appear in recent literature.

In 1952, Barker[1] presented before the New York Academy of Medicine the case of a woman with

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recurrent pretibial ulcerations in whose blood L.E. cells were found. In the same year, Schoch,[2] in describing leg ulcerations in Felty's syndrome, reported a patient whose bone marrow contained L.E. cells. Schoch mentioned the possibility that some cases of Felty's syndrome may, in fact, be instances of latent lupus erythematosus. Gaté, et al,[3] presented a patient with chronic arthritis and recurrent leg ulcers, the vascular changes of which were thought to resemble those of periarteritis nodosa. There was marked leukopenia, however, a finding more suggestive of lupus erythematosus than of periarteritis nodosa.

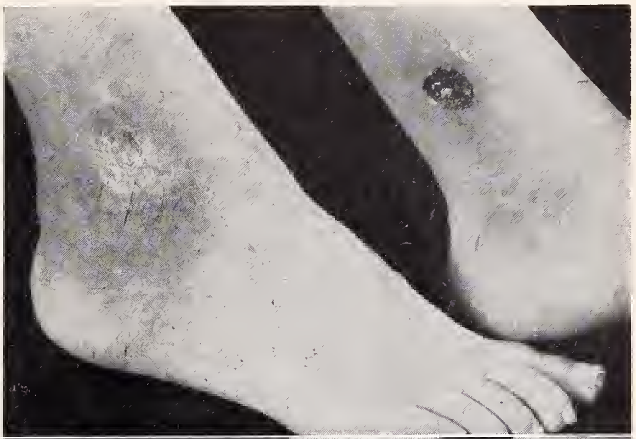
Early in 1957, Allison and Bettley,[4] under the title "Rheumatoid Arthritis with Chronic Leg Ulceration," reported six patients with arthritic symptoms and recurrent leg ulcers, some of which were strikingly similar to the ulcers in our patient. Four of these six patients were found to have positive tests for L.E. cells, and these authors commented on the strong possibility that the "rheumatoid" arthritis in these patients may have been part of the lupus erythematosus syndrome.

The reason for the appearance of these ulcers, and for their slowness in healing, remains unknown. Those we have observed in our patient, and others reported in the literature, resemble, in their early stages, localized infarctions of the skin and subcutaneous tissues. This suggests occlusion of some large arteriole, either by thrombosis or spasm; however, the failure of these ulcers to heal over long periods of time, and their rapid response to corticosteroids, suggest that some mechanism other than simple vascular occlusion is involved.

Unfortunaely, histologic study has given little information. Various vascular and connective tissue changes have been reported, but are probably secondary alterations. It would seem that any histologic examination short of serial sectioning of an entire lesion (and only in its early stages) would be quite meaningless. Unfortunately, such a complete study has not been practicable up to this time.

Summary and Conclusions

A patient with recurrent, slowly healing ulcerations of the lower legs, associated with long standing lupus erythematosus, is reported. Several similar cases have been found in the literature.



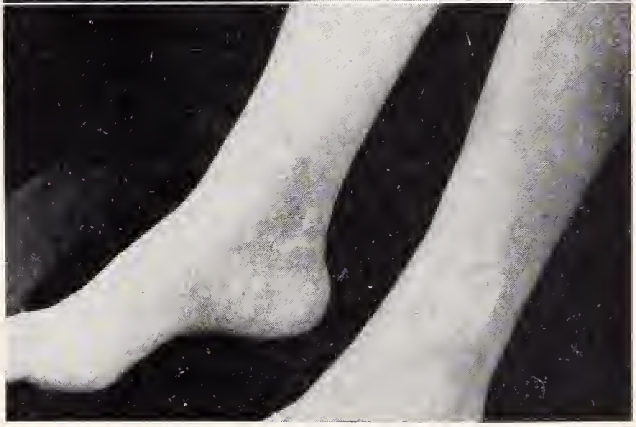


Fig. 1. (*above*) Old ulcer on lateral right ankle, and more recent ulcer on medial left ankle.

Fig. 2. (*below*) Numerous scars, marking sites of former leg ulcers.

After failure to respond to other therapeutic measures, rapid healing followed administration of corticosteroids.

It is believed that these lesions should be considered part of the syndrome of lupus erythematosus.

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Tumor Conference

Section Editor
CLAUDE HITCHCOCK, M.D.

CASE STUDY, MINNEAPOLIS GENERAL HOSPITAL

Pain in her back and hips caused the patient, aged sixty-five, to consult her physician four months prior to the time of her admission to the Minneapolis General Hospital. After several months' effort to establish a definitive etiology of the back pain, a mass was discovered in the right breast.

Examination at General Hospital revealed a bed-fast woman with no history of having previously been aware of the mass in the breast. During several months, the patient had lost between 35 and 40 pounds, and the pain in her back and hips had become progressively worse. Physical examination revealed a mass measuring 3 by 4.5 cm. in the outer upper quadrant of the right breast which was fixed to the underlying musculature. A large fixed nodule was present in the right axilla, but there were no palpable supraclavicular nodes.

The liver was slightly enlarged, but smooth and nontender. Blood studies revealed a normal hemoglobin and differential count. The patient had a few pus cells in the urine and a blood urea nitrogen of 11. The liver profile chemical studies were within normal limits. The alkaline phosphatase, however, was 26 King Armstrong units, representing a marked increase, and the acid phosphatase determination was 2.9 King Armstrong units. A serum calcium of 12 mg. per cent was found, with a phosphorus of 3.7 per cent.

Roentgen studies of the skeleton revealed multiple rib fractures and diffuse involvement of the skeletal system with osteoblastic and osteolytic metastases.

Upon admission to the hospital, the patient was taking premarin in a low dosage. This had been started several days prior to admission. Since this patient came to the hospital with probable breast carcinoma with widespread distant metastases, a program of hormone therapy was considered. The patient first had a single incisional biopsy of the mass in the right breast which proved to be a primary adenocarcinoma.

She was started on stilbestrol therapy, 5 mg. three times daily, and after several weeks a balance study of her calcium metabolism was undertaken. While on a daily total calcium intake of 200 mg. or less, the patient excreted from 109 to 277 mg. of calcium per day. After six weeks of stilbestrol therapy, the hormone was discontinued and one month later the calcium excretion studies revealed the daily urinary calcium to vary from 28 to 82 mg. (while the patient was on a daily diet of 200 mg. of calcium or less). It was the consensus of the staff that this patient responded in a negative manner to the estrogen therapy and this form of hormone treatment was contra-indicated. The patient then received x-ray therapy to the involved areas of weight bearing bones, and improved somewhat with relief of pain for a period of several months.

The patient re-entered the General Hospital during December, 1956, with increased pain and discomfort. A daily regimen of a 200 mg. calcium diet was started on December 28 and was continued for nine days. During this period, the urinary calcium excretions varied in the neighborhood of 76 mg. of calcium per day. Testosterone propionate in a dosage of 75 mg. was started on January 4, 1957, and was given twice a week until January 22. The patient received a total of six doses of testosterone propionate. The calcium balance studies were again started on January 17, 1957, and four determinations ranged from 87 mg. per day to 346 mg. per day. It appeared that this patient was again having an exacerbation of the bony metastases by the administration of testosterone propionate and therefore this hormone therapy was stopped on January 22.

It is well known that approximately 35 per cent of patients in the postmenopausal age group, who are placed on estrogen therapy for metastatic breast disease, will respond objectively to hormone therapy. It is also well established that about 25 to 30 per cent of patients with bony metastases will respond in an objective manner to estrogen therapy or to testosterone therapy. It is entirely conceivable that, on occasion, we can do harm to patients by administering hormone therapy when it actually has an adverse effect by fostering tumor growth. The use of calcium balance studies to determine the efficacy of hormone therapy can be recommended, although it is a costly and time-consuming procedure. Under the programs of charity type hospitals, however, where patients are hospitalized for prolonged periods of time, this program is feasible. The results in this patient would indicate that both estrogen therapy and testosterone propionate therapy were contra-indicated because of the unfavorable biologic characteristics of the metastatic breast cancer in this instance. The patient continued to worsen and subsequently died of the disease.

Editorials

John F. Briggs, M.D.
Arthur H. Wells, M.D.
Henry G. Moehring, M.D.

WMA AND WHO
The Difference

1. WMA is an organization of national medical associations. The unit of membership is the most representative national medical association in each country. It is completely non-governmental. It is not part of the U. N. It is a voluntary organization.
2. WMA represents the practicing medical profession.
3. WMA was organized in 1947 by AMA representatives and Western European medical leaders. Purpose was to exchange medical knowledge, to protect the freedom of medicine, and promote world peace.
4. Each member association sends two delegates, two alternate delegates and observers to the General Assemblies—the supreme policy making body of WMA.
5. The executive body of WMA is the Council. This meets twice a year and comprises 11 members elected from the Assembly and the President, President-Elect and Treasurer.
6. WMA is supported by members' dues and contributions and the annual budget is about $165,000.
7. American physicians and allied corporations interested in the work of WMA are organized as the United States Committee of The World Medical Association.

1. WHO is an intergovernmental health agency. The members are the governments that accept the nine principles upon which WHO is founded.
2. WHO represents governments in their public health and medical activities.
3. WHO is the result of proposal of U. N. in 1945 to create a specialized agency to deal with all matters related to health on a world-wide scale.
4. Each member government sends three delegates, chosen preferably from the national health administration of the government, to the annual World Health Assembly.
5. The Executive Board of WHO is the executive body and consists of 18 members elected to represent 18 member governments.
6. WHO is supported by dues allocated by the U. N. scale and the budget for 1958 is $13,000,000.
7. American citizens interested in the work of WHO are organized as the Citizens' Committee for the World Health Organization.

CLINICAL PSYCHOLOGY—HISTORY

The opportunity to state the case of clinical psychology is a welcome one. This editorial will deal with historical background. The next will explore the current status of the field, particularly in Minnesota. The last will involve guesses as to the future pattern of the profession.

The heritage of clinical psychology goes back over eighty years to the University of Leipzig and to Harvard in this country. Its parents were philosophy and physiology; and for an understanding of psychology today it is well to keep in mind that its birth, and most of its maturing process as well, took place within the academic setting. Scientific thought, inquiry, and experimentation were in the strict academic tradition; and contact with the medical school and sick people has been a development largely of the past two decades. Most of the earlier work (and much of the present) involved animal experimentation at a basic level. How the experimental animal work, researches in conditioning and learning, for example, might apply to suffering humanity, if it applied at all, was not a conspicuous preoccupation of psychology during its first half century. Thus the psychologist, until quite recently, was a university scholar with his office in or next to his laboratory which often contained an animal colony with much physical equipment. If he knew members of the clinical medical faculty of his university, it was usually through mutual interest in physiology, anatomy, or general biology. He would usually be well known in the university machine shop, for he often made much of his own equipment. These years laid a firm foundation for controlled experimentation and scientific tradition.

In view of such a background, how did the specialty of clinical psychology develop? This can be traced largely to that parent aspect of psychological research which related to the testing of intelligence. The early work of Binet and Simon led to standardized tests of intelligence, the scores of which were then related to chronological age to provide the intelligence quotient (I.Q.). As these tests came into use, it followed that they would be valuable in the diagnosis of mental deficiency and herein the psychologist began to associate with the physician. This first relationship occurred in the pediatric age group and hence the first "clinical" psychologist was found in institutions and clinics diagnosing or caring for children. In World War I derivatives of these tests were spectacularly applied for the screening of adult men. The various measures of intelligence are now so well established that it would be a poor children's clinic indeed that did not have such tests available to it. In any event, this development saw the birth of the "clinical" psychologist, i.e., the psychologist being used in the medical setting.

It was not a big step from intelligence tests to tests for personality, and this aspect of testing came into prominence, together with testing for aptitudes, during World War II. We can be proud of the fact that some of the significant research in personality testing was done at the University of Minnesota. Williamson, Paterson, Hathaway, and McKinley are names internationally famous in this field. The Air Force particularly was much interested and extensively used personality and aptitude tests in the selection of flying personnel. The Army and Navy did likewise for their people.

Thus the psychologist of today who does research, teaching, and service in the hospital and clinic traces his ancestry directly to the academic tradition; his intellectual heritage is a fusion of philosophy and physiology. His entrance into the clinical setting began with intelligence tests, standardized on "normals" but which found ready application in measuring the extremes of intelligence, i.e., the bright and the dull. In this context, the psychologist found himself working with physicians caring for children and aiding them in diagnosis. The "clinical" psychologist was born.

Donald W. Hastings, M.D.

ABOUT PRINTERS

> I suffered so much from printers' errors
> That death for me can hold no terrors.
> No doubt this stone has been mis-dated.
> I wish to Christ I had been cremated.
> (Daniel George's *New Epitaphs*, c. 1957)

This epitaph mildly expresses what any writer feels when he finds printers' errors in his deathless prose or poetry. Impatience with mistakes in printing quickly grows into bitter intolerance of the printer when the writer realizes how many thousand times the error was repeated and how permanently it stands there for everyone to see. Further fuel is added to the hot hate by the aforesaid everyone's happy eagerness to carry the error to the author and to remark cuttingly about it.

We plead for temperance toward the printer and cite these data in support of such moderation: Each of the letters, spaces, punctuation marks, et cetera in your masterly production reaches the printed page via a little piece of metal, maybe several pieces of metal—the type bar on the typewriter, the matrix in the typesetting machine, and the bits and squiggles inserted to maintain spaces, call attention to footnotes, and other pertinent information. This page, for example, involves at least 5000 such pieces of metal, some of them so small that they are handled with tweezers because fingers are too thick or clumsy. Not only must each of these bits of metal be in the right place at the right time in the process of getting this into print, but every time the type for the page is moved, the risk of disarranging some of them arises. And if the first setting of the type proves to have an error in it, the resetting to correct the error may also disarrange the metal pieces to yield a greater error than the one corrected.

No matter how much of their work printers try to reduce to foolproof typesetting, somewhere along the line you always come back to individual pieces of metal and thus printers remain craftsmen who, on each page of type, have over 5000 possibilities of making a mistake. Like us, fully aware that attaining perfection consistently is impossible, they nevertheless continue to strive to attain it. That they succeed so often in producing an errorless page is the wonder which you can afford to feed your outraged ego when you find a mistake in *your* article.

H.G.M.

MEDICAL LIBRARY SERVICES

Doctors who use medical libraries in this second half of the twentieth century have an assurance of what they want in the way of materials and of services from them. Through the growing adequacies of collections and services in medical school libraries, many physicians are graduated from their alma maters with definite concepts of what materials should be in the library, how they may be found, which includes short cuts, card catalogs, indexes and abstracts, and where they may be found.

All that a physician expects of a medical library, whether it is operated under federal, society, industry or hospital auspices, the librarian has the responsibility and the privilege of providing for him. He may then expect orientation to the particular library which he has selected to use. Again considering variations in individual libraries the new patron may be shown a floor plan of the library on which is indicated the library's important tools of communication, card catalog, indexes, abstracts and the location of reference, journal, reading room and stack divisions. A tour will lead him specifically to the locations of these important sources of information. Some explanations in detail may be in order. The dictionary catalog, which is the index to the library's complete holdings of books and periodicals, and especially if it varies in arrangement from the standard subject-author type, will be fully explained. If the student or practitioner has a limited knowledge of bibliographic tools, the underlying principles should be reviewed with him. Care must be taken that the use of such important indexes as the Current List of Medical Literature, whose entry forms deviate from other indexes, is understood. Any extra indexing project that the library carries should be pointed up as the important access to the current information actually in the library.

A potential client should be given a copy of the library's policies which informs him of the hours for service, the length of loans, the policy for borrowing books as supplementary material from other libraries. Also he will need to know if the library offers an extensive bibliographic service including translation and the editing of papers and where these may be obtained if the library does not provide them.

In a large library which will necessarily be supported with adequate funds, or nearly so, physicians may hope to find practically all their current reference wants. If the institution has a research program, the library will be geared to support it. Doctors are entitled to find in small libraries an adequate up to date working collection of standard textbooks and monographs in fields in which the institution does its major work. The larger the affiliating institution and the farther away the library is from larger collections in medicine, the more extensive its development must be in all subject fields. A medical library usually allocates two-thirds of its fund for the purchasing of current journals. This periodic material, because of the speed of scientific investigation, is every doctor's reading prerogative.

Another important, although possibly not essential, aspect of library service is pleasant surroundings. More and more libraries are moving into areas and furnishings conducive to relaxation and concentration. However, if a choice were to be made between a well-organized, carefully selected collection housed in gloomy, unattractive surroundings and a modern or elaborately furnished room with a meager collection of out-dated books, the preference would be surely for the former. Fortunately, libraries are keeping pace with modern trends in architecture and furnishings. Acoustic tiles and comfort cooling are making almost any location acceptable for the library, providing it is reasonably convenient to the main streams of traffic within the institution. Adequate lighting, comfortable chairs and reading lamps, private study units, or carrells, and open stacks for browsing rank high as aids to study, for practitioner, student and research worker alike.

Within this world of "miles of print" the difference is not so much whether the collection is large or small, clinically practical or directed toward research, as how it is organized and administered. This reverts again to the human components in library service, the librarian and her staff. If a poll were taken to evaluate the effective services of any medical library, the librarian's friendly, willing and co-operative attitude might rank high on the scale of appraisal as indicative of what doctors want, expect and really need from their libraries.

Sister Teresa Louise
Medical Librarian

LIFE INSURANCE IN ESTATE PLANNING TODAY—III

Should Life Insurance Policies Play a Bigger Role in Your Estate?

Under the new tax law, it is possible to avoid estate tax on the proceeds of policies taken out on your life even though you pay all the premiums. The basic requirements are: (1) the proceeds must be payable to a beneficiary other than your executor or your estate; (2) you must have no strings on the policy that the law will view as incidents of ownership.

These requirements would be met, for example, by naming your wife or child as beneficiary, and transferring the policy with all incidents of ownership to either the beneficiary or a trustee.

The only estate tax danger in this arrangement lies in the possibility that the Treasury will claim that you transferred the policy or paid the premiums "in contemplation of death." But this danger is completely removed if you live more than three years after the policy transfer and the last premium payment. And even if you were not to live three years after the last premium payment, the probability is that most of the proceeds would escape estate tax.

When *new* insurance is purchased and the wife is the applicant, the owner and the beneficiary, the "contemplation of death" provision no longer applies.

Note that you can avoid gift tax on transfers of policies through use of the $30,000 over-all exemption and the $3,000 annual exclusion. And if you are married, you can transfer free of gift tax to one beneficiary a partly or fully paid-up policy worth up to $66,000. Payment of premiums on a transferred policy would also be tax-free up to $6,000 annually (plus any additional premiums for which the over-all exemption is used). The policy does not have to have a cash surrender value to qualify for the annual exclusion.

Should Income Shifting Play a Part in Your Estate?

A means of achieving a "triple tax" saving (Income, Estate and Inheritance) is the use of the Short Term Trust, using the trust income to purchase life insurance on the life of the mother or on the children themselves. The father sets aside some securities or property, and has the income therefrom payable to a trust, administered by a disinterested trustee. The trustee uses the trust income to purchase life insurance policies on the lives of the children. The trust may be for the life of the beneficiary or for any period ten years or over.

So we find a four-way tax saving:

1. The trust pays the tax on the income received in the trust shifting the tax out of the father's high bracket to the lower bracket of the trust.
2. If the grantor's wife is named beneficiary of the life policies on the child, and the child dies during the trust period, $4,000 of the proceeds pass free of state inheritance taxes.
3. If the trust income is less than $675 to each child, or the child is under 19 or a student and receives over half of his support from his father, then the grantor will not lose the tax exemptions. The trust also receives a $100 tax exemption in and of itself.
4. If the grantor dies while the trust is in operation, a certain portion of his estate has been cut away with a subsequent lower estate tax.

Three items you should be careful about; the trust must be set up for ten years or more, or for the life of the beneficiary and the grantor must not be the insured, and the trust income should not be used for support of the child which would make the trust income taxable to the grantor.

Alan W. Giles

THE USES OF WAXED PAPER FROM A MEDICAL STANDPOINT

Most of us think of waxed paper in terms of the familiar household roll that is so handy for everyday kitchen and household chores. But the greatest use of waxed paper in today's living is as a flexible packaging material that offers many outstanding advantages to its users.

Waxed paper is a leading package wrap for most processed foods and food products, those items which are of vital concern to the health and well-being of everyone. It provides full flavor and freshness protection, along with established sanitation "plusses."

Bread and other baked goods products are most often packaged in waxed paper. Indeed, 82 per cent of all white enriched bread is waxed paper-wrapped, as are many of the specialty health, vitamin and protein breads that are of growing popularity in people's diets. In addition, most crackers, biscuits and cereals are wrapped in waxed paper, or packaged with waxed paper or waxed glassine inner wraps. Even butter and other dairy products are being packaged more and more in waxed paper, for extra protection.

Another leading food use of waxed paper is in the packaging of frozen foods. With the particular problems of frozen foods, such as changes in water content, et cetera, the "ideal" package must have certain characteristics to fully protect the product and prevent foods from spoiling before use. Waxed paper is such an "ideal" package.

Because of its low water vapor and oxygen permeability, waxed paper serves as a backstop against the passage of liquids. It is impermeable to fats and oils, resists acids and other corrosive food components. Moreover, waxed paper keeps frozen foods palatable by excluding foreign odors and preventing the transfer of food flavor.

Perhaps most important from a medical point-of-view, waxed paper prevents light degradation of vitamins and enables frozen foods to be delivered to their ultimate user in the most healthful and health-giving form. Similar advantages accrue in the use of heavily waxed, opaque locker paper for storage of foods in home freezers.

Hospitals and other institutions greatly benefit from the widespread use of waxed paper in packaging food products. In the first place, foods stay fresh longer and there is less spoilage, which holds down operating costs due to food waste. Moreover, waxed paper enables a dietary portion control program that saves money for the hospital and makes for happier, more satisfied patients.

Indeed, hospitals can offer more varied and interesting menus to their patients through tastier, easy-to-prepare foods packaged in waxed paper. The convenience factor, along with elimination of waste and morale-building aspects, all combine to enable the hospital to play its part in healing the sick.

But food use of waxed paper is only one of many ways in which waxed paper is of help to doctors and hospitals in today's modern usage. Other outstanding uses of waxed paper, keyed specifically to the medical profession, will be explored in a second discussion.

Waxed paper, which plays its largest role in today's living as a leading packaging material for bread, cereal, frozen foods and other processed food products, nevertheless has many outstanding modern uses that are keyed specifically to the medical profession.

Doctors and nurses, as well as hospitals themselves, put waxed paper to work for them in many ways. Certain technical qualities and physical properties of waxed paper make it especially useful in the medical field. One example might be its highly waxed finish that prevents other substances from adhering to waxed paper; in fact, waxed paper sticks solely to itself, and then only when it is heat sealed to form a strong bond.

Looking over the field of hospital operations, we find that waxed paper often is used to store the plaster from which casts are made. Similarly, pharmacies, whether or not they actually are located in hospitals, use waxed paper in filling prescriptions. They place it on scales to serve as a base for weighing powders or ointments, and the waxed paper keeps the scale clean. When used as a base under the scale itself, waxed paper serves to keep the counter clean as well.

Surgery in modern-day hospitals also finds a number of uses for waxed paper. It serves as a temporary wrapping for tissues sent from the operating room for testing in the pathology laboratory. Moreover, waxed paper often is used to preserve different kinds of physical specimens, and as a cover for wet gauze.

Wet dressings, whether they are applied in a hospital, the doctor's office or at home, benefit from the use of waxed paper. It holds in the moisture and prevents the bandage from wetting the bed clothes or the patient's clothing. This is the case for such widely divergent undertakings as treatment for burns or wet packs for physical therapy.

Of course, waxed paper plays another role in medication that often is overlooked. Most bandages, surgical dressings and sterilized gauze are packaged in waxed paper by the manufacturer for sanitation and cleanliness. Some doctors even protect their instruments after sterilizing them by covering or wrapping them with waxed paper.

Kitchen uses of waxed paper are limitless, and the busy hospital kitchen preparing meals for hundreds of patients finds waxed paper just as useful as does the housewife making dinner for her family of four. Only the degree is different!

The big advantage that waxed paper offers to hospital kitchens is that it enables them to prepare special diets for patients beforehand and keep the food stored until mealtime distribution. The food remains fresh and flavorful; it is kept completely sanitary. In addition, nourishment "pick-ups" for patients, like glasses of fruit juice, et cetera, can be covered with waxed paper and stacked on trays to be passed out as requested or prescribed.

These are but a few of the ways that waxed paper through its modern usage plays a vital but often unrecognized role in today's medical picture. Waxed paper's versatility and adaptability truly enable it to make a marked contribution to the health and well-being of everyone.

John A. Egan

THE TUBERCULIN TEST

When Koch announced the discovery of the tubercle bacillus in 1882, the first specific evidence in the diagnosis of tuberculosis became available; namely, the recovery of tubercle bacilli from secreta or excreta of persons who had tuberculosis. The same Koch announced the preparation of the second specific agent in diagnosis; namely, tuberculin, in 1890. He and his immediate disciples first used this substance as a therapeutic and preventive agent for each of which it failed. However, they observed that when introduced into the bodies of persons who had tuberculosis, local, focal and constitutional, reactions occurred which were helpful in diagnosis when this disease was suspected.

Koch introduced tuberculin subcutaneously, but his dosage was so large and reactions often were so severe as to cause harm. Veterinarians in various parts of the world including Leonard Pearson and Charles Cotton of the United States tested large numbers of cattle. At necropsy, lesions were never found in non-reactors, but were nearly always in evidence in reactors, regardless of the previous health status of the animals.

Dr. Talbot Jones, St. Paul, was in Berlin when Koch announced that he had prepared tuberculin. In 1891, Doctor Jones said that the time would probably come when the physician who failed to administer the tuberculin test would lay himself open to just censure.

Apparently, Dr. W. J. Mayo was the first to use tuberculin as a diagnostic agent in Minnesota. In 1894, he said that he had used it when it was difficult to determine the exact nature of disease and had found that it would almost invariably lead to the correct diagnosis. In 1896, Dr. E. J. Davis, St. Paul, stated that tuberculin might do much toward checking the spread of "the great white scourge."

From September to December, 1894, Dr. C. N. Hewitt, Executive Secretary, Minnesota State Board of Health, personally directed tuberculin testing by Dr. Charles E. Cotton of 335 cattle. By December 31 of that year, forty-one of the reactors had been slaughtered and the post-mortem examination sustained the diagnosis. Veterinarians throughout the nation adopted the tuberculin test as their sole diagnostic agent in a national tuberculosis eradication campaign among animals. From 1917 through the fiscal year 1957, veterinarians of the United States alone did post-mortem examinations on 4,062,634 cattle slaughtered because they reacted to tuberculin. This has thoroughly convinced well-informed veterinarians that a characteristic tuberculin reaction practically always means the presence of at least microscopic tuberculous lesions.

Beginning in 1907, Pirquet reported a testing technique which consisted of applying tuberculin to a superficial skin scarification. A year later, Mantoux introduced the intracutaneous method of administration and the same year Lautier reported observations on applying tuberculin directly to unabraded cleansed skin. This method was later revived and is now known as the patch test.

Each of the numerous methods of administration has had its disciples and each one has had its pros and cons. At the present moment, the intracutaneous method (Mantoux) is generally considered to have superiority over the others. This does not mean that other methods are not useful or that they should not be employed when conditions warrant.

From 1908 to 1912, Ghon made meticulous post-mortem examinations of the bodies of 184 children who during life presented no evidence of tuberculosis except the tuberculin reaction. The main object of this study was to determine whether the tuberculin reaction always indicates the presence of tuberculosis. He found the disease in all but one which was not completely examined. When his first examination failed, he took a second, and sometimes a third look before finding the lesions because of their minuteness or location. In five of the bodies, the lesions were located extra-thoracicly. Following this, Ghon said, "From the standpoint of the pathological anatomist, my researches, which in regard to the question are not limited to the cases described here, justify me in concurring entirely with those who advocate the specific value of the tuberculin reaction."

There are two tuberculin preparations now in extensive use, namely, Koch's original tuberculin (OT) and purified protein derivative (PPD). From the standpoint of eliciting sensitivity of tissues they are approximately equal. Each has its advantages and disadvantages, but a characteristic reaction from either one indicates the presence of tuberculosis in some stage of its development from microscopic to gross lesions. Therefore a tuberculin reaction justifies the diagnosis of at least the primary type of tuberculosis. From the reaction there is no way of determining, however, whether the reinfection or clinical type of disease has or will develop. If the person who reacts to tuberculin has a clear x-ray film, as is true in most cases on first examination, and has no other evidence of the disease, he should be examined periodically throughout life. This procedure will detect those chronic clinical lesions destined to develop on an average of two or more years before symptoms appear and when treatment is most effective.

Since morbidity and mortality from tuberculosis have so decreased in Minnesota, one frequently hears physicians state that they have not seen a case in a year or more. They are referring to gross lesions detectable by x-ray shadows, et cetera. In all probability such physicians have cases in their offices nearly every day who have earlier stages of the disease which can be detected only by the tuberculin test. Every such person should be advised of this condition so as to be on the alert for possible subsequent clinical disease.

Persons who do not react to tuberculin should have the test repeated periodically at least once a year. The person who has recently become a tuberculin reactor, has usually been in direct contact with a contagious case of tuberculosis since the last negative test. Seeking that contagious case among the adult contacts of the recent tuberculin converter is an excellent case-finding method.

The magnitude of the tuberculosis problem in any family, community, county or state can be determined only by testing all persons with tuberculin. The problem lies among those who react, since each reactor who does not already have clinical disease is a potential case at some subsequent time. Information obtained in Kittson County, Minnesota, is a good example. In 1955 only one person died from tuberculosis in that county and only three new clinical cases were reported. However, when the tuberculin test was offered to the entire citizenry, approximately 2100 reacted. For the most part, these reactors were 40 years of age or older. They were persons who had not been protected against infection during childhood and early adulthood. Among these 2100 persons clinical disease will evolve in a considerable number as the years pass. Similar observations have now been made in several Minnesota counties and municipalities with analogous results, as reported by Jordan and Jordan and Billyeald.

The tuberculin test is the only method by which responsibility can be accurately determined in cases that come to litigation. It is the place where the infection was allowed to occur as indicated by the tuberculin reaction that is responsible rather than the place where the individual happens to be when clinical disease evolves to demonstrable proportions.

Now that antituberculosis drug administration is recommended in many places as soon as possible after tuberculous infection occurs, the importance of periodic tuberculin testing is obvious. In the recent tuberculin converter, lesions are usually still microscopic and vascular so that drugs may be expected to reach all tubercle bacilli. Although present drugs only suppress them, there is hope that a thoroughly germicidal drug will soon become available which will sterilize such lesions. If tuberculosis is not found in this early stage, the lesions may lose their blood supply and there is little likelihood that a germicidal drug in high concentration in the blood stream could reach the bacilli in avascular necrotic areas.

Testing with tuberculin is the best method of disseminating information about tuberculosis that has ever been devised. This applies to the individual, family and the public.

J. A. Myers, M.D.

President's Letter

A CENTENNIAL YEAR INVITATION CORDIALLY EXTENDED

This is a particular, special personal invitation to all members of the Minnesota State Medical Association to come to the Annual Meeting of the Association. There are changes this year. The meeting is in the last half of the week instead of the first half. There are more symposia and panel discussions. The scientific exhibit is larger than it has ever been. The program is outlined elsewhere in this issue of Minnesota Medicine.

Because the World Health Organization meets in Minneapolis from May 26 to June 14, 1958, immediately after our meeting, we have been able to devote Friday afternoon to virus diseases and to a report of WHO studies around the world. Dr. Robert Barr has obtained for us Dr. Kleinman from the State Board of Health to discuss current virus disease problems and Dr. Mauricio da Silva of the Pan American Sanitary Bureau to report on immunization with orally administered live virus poliomyelitis vaccine. Dr. da Silva's work was done here and has been continued since he left here. Dr. Fred L. Soper, the director of the Pan American Sanitary Bureau, will discuss these problems. The Pan American Sanitary Bureau is the Western Hemisphere department of the WHO.

The last half of this afternoon will be occupied with World Health Organization Studies and Reports by Dr. Marcolino G. Candau, the Director General of WHO with headquarters in Geneva, Switzerland. The moderator for this session will be Dr. Walter Judd and the discussion will be by Dr. Leroy Burney, Surgeon General of the United States Public Health Service. The principal speaker at the annual banquet will be Dr. David Allman, the President of the American Medical Association.

For three weeks Minneapolis will be literally the public health center of the world. The representatives of 87 countries will be here for the WHO. We who have the good fortune to live in Minnesota have always thought our State to be beautiful and, in many ways, important. Probably we have never even thought of anyone from India, Russia or Africa knowing anything about us. Now we hear that Public Health Officials all over the world are striving for the opportunity to come here. Minneapolis was selected by the WHO for this tenth anniversary meeting partly because the facilities are here, but more, because Minnesota and the midwest are more typical of the real United States than are the large seaboard cities.

We hope that you will come to the 105th annual meeting of the Minnesota State Medical Association and that you will stay for, at least, part of the World Health Organization. We hope that you will meet the visitors from foreign countries and that they will meet you. And we hope our own meeting will be valuable and enjoyable. We hope also that you will listen in on the House of Delegates meetings and that when you leave you will be well informed medically and will know more about the problems facing medicine as a whole.

Horatio B Sweetser

President, Minnesota State
Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

CRUISE DEDUCTIONS QUESTIONED

Recently, a San Francisco Tax Court upheld a Tax Commissioner's action which disallowed 80 per cent of the cost of a physician's cruise, holding that the major portion of the cruise was incurred primarily for a vacation—a non-deductible item.

The cruise sponsored by the American College of Physicians included a series of scientific discussions in addition to sightseeing. The court held that the technical sessions were of short duration.

The proportionate allocation was upheld because the taxpayer physician failed to show how his time was budgeted. Tax authorities did not question the physician's right to deduct the entire cost of attending a convention held prior to the cruise as a business expense.

UNCLE SAM—MEDICALLY SPEAKING

Recently released transcripts of testimony on 1958-59 appropriations for the Department of Health, Education, and Welfare indicate the increasing role of government participation in providing medical care for the American public. Activities included in the hearings are considered vitally important to this nation's practicing physicians. Following are some of the highlights:

Health Insurance

Passage of a law permitting smaller underwriters to pool risks in order to provide coverage for older persons and farm persons is being urged by Secretary of HEW, Marion B. Folsum.

Quackery

The Food and Drug Administration has increased the tempo of its activities to combat the twentieth century methods used by the quack to bilk millions of dollars from the American people by fraudulent means.

Disability Benefits

Approximately 160,000 persons have taken advantage of Social Security disability cash benefits during the first six months of the program's existence. Five-month medical determinations of disability have been cut to three. More than $2 million will be spent in the year beginning July 1, 1958, for purposes of examining applicants for disability benefits.

Illicit Drug Sales

Certain Texas and Georgia physicians have been under fire from Federal Authorities for dispensing abnormally large quantities of amphetamines. The report cited one case where an under cover agent purchased 55,000 tablets from one doctor over a period of twenty-eight days.

Hospital Construction

House approval of more than the $75 million requested by President Eisenhower for Hill-Burton grants in aid to help finance construction and improvement of hospitals throughout the country is expected to receive favorable attention.

THIS MATTER OF GIVING

Americans give an estimated annual $6 billion to charitable and religious organizations. Some would say this is but a fraction of what Americans could give if they but would.

All about us are evidences of benefits derived through American benevolence. Conversely, there are untold instances of the marks of the charitable charlatan. Giving ought to be a personal matter—not brought about by habit or high pressure.

Who should receive how much and for what purpose should be individually determined.

When in question about the good intentions of a potential recipient, check with the local Community Chest, Better Business Bureau, Health Agencies, Chamber of Commerce, Bankers or local Clergy representatives.

When you wish to give:

1. Give only after you investigate the cause, the sponsor and the purpose for which the funds will be spent.
2. Give objectively—don't allow yourself to become carried away by an emotional "pitch."
3. Give only after you are able to identify patrons of the drive. "Director," "Coordinator," "Civic Leader" and other impressive titles can throw a left curve.

4. Give only if a telephone solicitor will fully identify himself, presenting all the facts.

5. Give only when a solicitor doesn't mind receiving your check via U. S. mail. Be on guard when a cash donation is requested.

6. Give only to street solicitors with cup, container or coin bank, when they represent an accepted cause.

7. Give only if the cause is recognized by the state law.

8. Give out of the goodness of your heart. Don't let friends or acquaintances apply "gentle pressure."

AMA PRESIDENT LAUNCHES ASSAULT ON FORAND BILL

A letter from the AMA President, Dr. David Allman, to approximately 140,000 doctors has urged a gross-root offensive to insure defeat of the Forand Bill. The letter urging defeat of the successor to the Wagner-Murray-Dingell Bill was brought to the attention of doctors at a time when congressmen and senators were home during the Easter holiday, sounding out public opinion.

Minnesota Representative, John A. Blatnik, is the latest to join the Democratic bloc of co-sponsors of the Forand Bill.

Senator Wayne Morse, Democrat from Oregon, recently made a floor speech urging enactment of Companion Bill (S-3508) to the Forand Bill.

"I want to express my personal thanks and appreciation," said Senator Morse, "to Mr. Forand for his leadership on this matter. I am happy to acknowledge that I have simply followed his lead by incorporating into my bill the health insurance program he had first introduced in HR 9467.

"I hope that, by so doing, I can reenforce the chances that a health insurance program will soon be adopted," stated Senator Morse.

Senator Morse's bill would amend the Social Security act (a) to provide hospital and nursing home care and surgical benefits identical with those in the Forand bill, (b) to increase the wage base and rate for social security payroll taxes and (c) to increase social security benefits. By substituting a table for the present law's formula to determine benefits, this bill would increase the minimum to $40 per month, the maximum family benefit from $200 to $346.40, and the maximum individual primary benefit from the present $108.50 to $173.20. Also, a more liberal "drop out period" would be provided. The wage base on which tax is levied would be increased from the present $4,200 to $6,000, and the rate increased to 3½ per cent for employer and employee and 5¼ per cent for self-employed for the 1959-60 period. By 1975, the respective rates would be 5½ per cent and 8¼ per cent. The bill also would increase U. S. public assistance payments by 25 per cent.

WELFARE BILLS CONCERNING THE AGED

In addition, the House Education and Labor Committee has conducted hearings on eighteen bills dealing with welfare of the aged.

Worth noting are bills sponsored by Representative Edith Green (D., Oregon) and John E. Fogarty (D., R. I.). Mrs. Green not only indorsed Forand bill but said legislation that would go further is needed. She states Government should make provision for defraying medical expenses of elderly persons who are *not* eligible for such protection under old age and survivors insurance. She also urged consideration be given the establishment of a separate institute on geriatrics in National Institutes of Health.

Her bill, along with ten other pending bills, would set up a bureau of Older Persons in Department of HEW. The Fogarty bill (HR 9822) proposes a White House conference on Aging, to be preceded by warmup conferences in the several states.

HEW SPENDING URGED

Government spending for medical research facilities, hospitals and water treatment plants from now to the end of the fiscal year ending June 30 are expected to exceed $800 million. Originally, appropriations for this purpose had been pared to $670 million. The increased spending by the Department of Health, Education and Welfare is an anti-recession measure designated to stimulate business activity.

Largest increase is in the building of medical research facilities by hospitals, medical schools and other institutions, for which the U. S. provides $30 million a year. Total value of contracts to be let in the period has been increased from an estimated $120 million to $182 million. Hospital contracts to be let under Hill-Burton will total $405 million, rather than the originally planned

$381 million, and sewage plant contracts will move up from $170 million to $215 million. In all cases, totals include local as well as U. S. money.

Rural Clinics Grants.—Senators Payne (R., Me.) and Flanders (R., Vt.) are sponsoring an amendment to Hill-Burton to allow private, non-profit associations or corporations to get up to $25,000 in federal funds on a matching basis for building and equipping diagnostic and treatment centers. Rural communities with population up to 15,000 would be eligible and the group need not be affiliated with a hospital as required for other Hill-Burton projects. This bill *(S. 3588)* was followed by one from Rep. Coffin (D., Me.) along similar lines. His bill, H.R. 11826, offers grants to a single town of not over 10,000 or a group of towns with no more than 15,000. One major difference with the Payne-Flanders bill is that in *H.R. 11826* the association or corporation would have to have a formal affiliation with a non-profit teaching hospital.

RECOMMENDATION FOR NURSING HOMES AND HOMES FOR THE AGED

Thousands of old people are not receiving even routine medical supervision in many of this nation's rest homes, much less, the benefits of recent advances in medicine of special value for severely handicapped patients. Secretary of Health, Education and Welfare, Marion B. Folsom, recently told representatives of approximately 150 individual homes, social service agencies, State Health and Welfare Departments and delegates from 32 national organizations. Twenty-eight states were represented. Secretary Folsom's remarks were made on the occasion of the first National conference on nursing homes and homes for the aged, which was held recently in Washington, D. C.

Surgeon General Leroy E. Burney stressed the lack of high-quality institutions in his address of thousands of older patients in general hospitals for longer than necessary, and deprives hundreds of thousands of other individuals of good care.

However, Dr. Burney emphasized, many institutions have demonstrated that the same great advances in medicine, psychiatry, sociology, architecture, construction, equipment and personal services which revolutionized the community general hospital are applicable to the nursing home and home for the aged. He said the main task of the Conference was to find ways that will enable all such institutions to achieve comparable results.

Following are facts about nursing homes and homes for the aged in the United States:

In the United States, there are about 25,000 nursing homes, with 450,000 beds. Of these beds, 180,000 are in "skilled nursing" homes and 80,000 in homes that provide skilled nursing care.

Eighty years is the average age of persons living in nursing homes. Two-thirds of them are over seventy-five years old, and two-thirds are women. Less than half can walk alone. More than half are disoriented at times. About half are incontinent, and two-thirds have some type of circulatory disorder.

Only 3 per cent of nursing homes are publicly owned. Seventy-one per cent of the beds are in private homes and 15 per cent in publicly owned homes. Privately owned nursing homes average about eighteen beds in size. Publicly owned homes are bigger, averaging about sixty-nine beds.

The average monthly charge for nursing homes care is $150. In thirteen states studied, the average cost of nursing home care was from $90 to $200 per month. Public funds pay for about half the people in nursing homes. Public assistance payments are from $55 to $155 per month.

Every state and territory, except the Virgin Islands and Puerto Rico, licenses nursing homes. Homes for the aged are licensed except in South Carolina, the Virgin Islands and Puerto Rico.

Responsibility for licensure is as follows:

	Nursing Homes	*Homes for the Aged*
State Health Dept's	42	34
State Welfare Dept's	6	14
Other State Agencies	3	2
None	2	3
TOTAL	53	53

Council for Health Care of the Aged Formed

The American Dental Association, the American Hospital Association, the American Medical Association, and the American Nursing Home Association have announced the establishment of the Joint Council to Improve the Health Care of the Aged.

Objectives of the council, the formation of which has been under consideration for some time by the sponsoring groups, are: (1) to identify and analyze the health needs of the aged; (2) to appraise available health resources for the aged; (3) to develop programs to foster the best possible health care for the aged regardless of their economic status.

The Joint Council to Improve the Health Care of the Aged is made up of three representatives of each sponsoring organization. One of the first jobs of the council will be to determine exactly what are the health problems of the aged.

The need for new programs in this field is accented by the fact that the life expectancy of individuals has been constantly increasing in recent years. In 1935, life expectancy in the United States was an average sixty years. The most recent figure indicates the average life expectancy now to be seventy years.

One of the principal immediate projects of the council will be the development of programs and facilities to be tailored to the health needs and finances of the aged.

Another facet of the council's broad-range program will be to work closely with health insurance groups in an effort to improve the coverage of the aged and to see that their insurance dollars go further.

It is the belief of the Joint Council to Improve the Health Care of the Aged that much can be done for older people by the states and communities, and the council will endeavor to stimulate the activities at these levels of government.

Special research projects contemplated by each of the organizations supporting the council will be pooled and programs developed to meet the health needs of the aged. The ultimate goal is to provide adequate health care at reasonable costs.

GOVERNMENT-SUPPORTED COLLEGE RESEARCH

Almost two-thirds of all money for research and development work at colleges and universities in the United States is allocated by the Federal Government according to a National Science Foundation survey. The extent of United States Government participation amounts to about $500 million. Most of the funds are earmarked for defense or similar projects.

PANCREAS AND DIABETES MELLITUS IN MAN AND ANIMALS

(Continued from Page 346)

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RUPTURE OF VAGINAL VARICOSITIES

(Continued from Page 347)

Observations

1. This case points out the importance of routine presurgical vaginal (manual and speculum) examinations on contemplated emergency Cesarian sections.

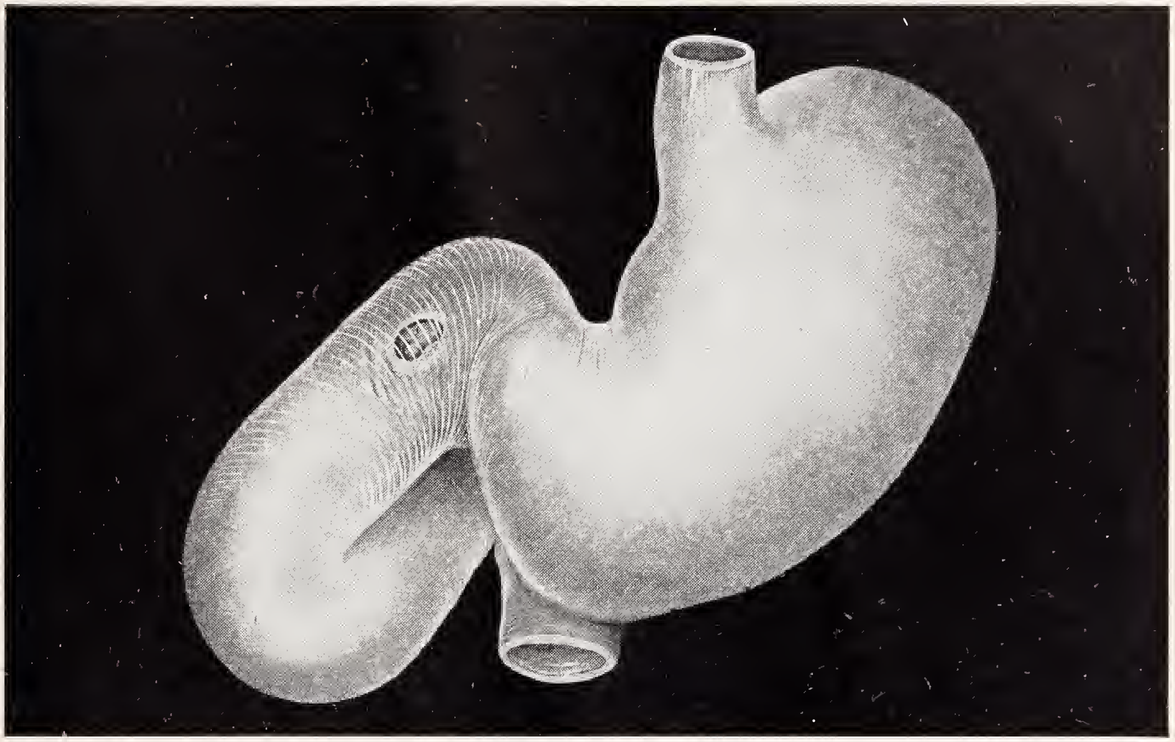
(a) In an emergency such as this, a Cesarian section might have been done in haste, on the presumptive diagnosis of placenta previa, with possible maternal exsanguination during the procedure at worst, and, at least, a resultant premature child, less qualified for survival.

(b) Vaginal examination may supplant, augment, or corroborate the results of x-ray in cases where shock is impending and definitive diagnostic measures are limited by the extremity of the patient's condition.

2. This case again stresses the important fact that there are causes of severe antepartum bleeding in addition to placental abnormalities.

3. A third point is that conditions not present in early pregnancy may develop at any time and cause complications in the last trimester.

CONFIRMED THERAPEUTIC UTILITY



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Therapeutic utility and effectiveness of Pro-Banthīne in the treatment of peptic ulcer are repeatedly referred to in the recent medical literature.

Pro-Banthīne Dosage

The average adult oral dosage of Pro-Banthīne is one tablet (15 mg.) with meals and two tablets at bedtime.

G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

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*Lichstein, J.; Morehouse, M. G., and Osmon, K. L.: Pro-Banthīne in the Treatment of Peptic Ulcer. A Clinical Evaluation with Gastric Secretory, Motility and Gastroscopic Studies. Report of 60 Cases, Am. J. M. Sc. *232:*156 (Aug.) 1956.

SEARLE

Increased incidence and increased utilization of the Blue Shield contract by participant subscribers during the year 1957 as compared to 1956 is shown by a comparison of the number of services of physicians provided Blue Shield benefits per year per 1,000 contracts and per year per 1,000 subscribers.

During 1957, Blue Shield provided benefits for 963 physicans' services per 1,000 contracts, or 362 services for 1,000 participant subscribers. In the previous year, 1956, Blue Shield benefits were provided for 859 services of physicians per 1,000 contracts, and 323 services per 1,000 subscribers.

The 1957 ratio of physicians' services provided Blue Shield benefits per 1,000 contracts and subscribers shows an average of almost one service for each contract in effect and over one service for every three participant subscribers.

Percentagewise, the increase in the number of doctors' services provided Blue Shield benefits per 1,000 subscribers and per 1,000 contracts in 1957 over 1956 is approximately 12 per cent. This percentage increase clearly illustrates the increased incidence and utilization experienced by Blue Shield in 1957 over 1956.

During 1957, a total of 307,171 services of doctors were provided Blue Shield benefits, while during 1956, Blue Shield provided benefits for 250,600 physicians' services rendered subscribers. The number of services for which Blue Shield provided benefits in 1957 is approximately 22 per cent greater than the number of services provided benefits during 1956.

Blue Cross subscriber usage of benefits is continuing at the high rate experienced during the last quarter of 1957. During the first two months of 1958, the 35,080 subscribers hospitalized represented 495 cases paid per year per 1,000 contracts protected, an increase of 3.8 per cent over the 477 cases experienced during the similar period of 1957.

The major portion of this increased usage, 83.3 per cent, is attributable to the increase in number of Blue Cross subscribers receiving hospital care for accidental injuries and poisonings. During the first two months of 1958, 6,257 subscribers representing 17.8 per cent of all Blue Cross cases paid received hospital care because of accidental injuries. During the same period of the previous year, 5,058 subscribers or 15.3 per cent of total cases paid were hospitalized due to accidental injuries. Blue Cross benefits for treatment of accidents incurred by subscribers averaged over $200,000 per month during the first two months of the year. Annually this would amount to $2,400,000 of Blue Cross benefits—or over $2 per person; man, woman and child, covered by Blue Cross in Minnesota—for hospital treatment of accidental injuries and poisonings.

The frequency of diseases of the digestive system incurred by Blue Cross subscribers to date this year as compared to the same period of 1957 is the second leading reason for increased usage of Blue Cross benefits during this period. During the first two months of 1958 over $900,000 in benefits has been provided 4,754 subscribers hospitalized because of diseases of the digestive system compared to approximately $780,000 in benefits provided 4,415 subscribers during the same period of 1957.

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BROAD THERAPY . . . The combined antibacterial effectiveness of neomycin and Sulfasuxidine is concentrated in the bowel since the absorption of both agents is negligible.

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Meetings and Announcements

STATE

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

NATIONAL

American Congress of Physical Medicine and Rehabilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

American Goiter Association, 1958 meeting, June 17-19, San Francisco, California.

Gerontological Society, Inc., Bellevue Stratford Hotel, eleventh annual scientific meeting, Philadelphia, Pennsylvania, November 6, 7, and 8, 1958.

Trudeau School of Tuberculosis and Other Pulmonary Diseases, forty-third session, Saranac Lake, N. Y., June 2 to 20, 1958. Detailed information may be obtained from the Secretary, Trudeau School of Tuberculosis and Other Pulmonary Diseases, Box 500 Saranac Lake, N. Y.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy, Association, Paris, France, October 19-26, 1958.

World Congress of Gastroenterology, Washington, D. C., May 24-31, 1958. Joint meeting with the American Gastroenterological Association, Dr. H. M. Pollard, Secretary-General, University Hospital, Ann Arbor, Michigan.

World Health Organization. Municipal Auditorium, Minneapolis, Minnesota, May 26-June 14, 1958.

STATE MEETING HAS CENTENNIAL "WHO" FLAVOR

The 105th annual meeting of the Minnesota State Medical Association will be held May 22, 23. and 24 in the Minneapolis Auditorium with headquarters in the Hotel Leamington. Association members are asked to note the change from a "week end" to a "middle of the week" opening day.

Delegates from eighty-eight nations of WHO, including the Minister of National Director of Health from each nation are expected to attend. Invitations have been extended to WHO visitors to participate in the various centennial program activities of the Minnesota State Medical Association. The program committee for the annual meeting has been fortunate in securing a varied program of speakers representing world renowned men from the areas of medical science, medical economics and world health.

A representative sampling from the speakers program includes: Mauicio M. da Silva, M.D., Pan American Sanitary Bureau, Washington, D.C.; Fred L. Soper, M.D., Director, Pan American Sanitary Bureau, Washington, D. C.; Leroy E. Burney, M.D., Surgeon General, Public Health Service, Washington, D. C.; Marcolino G. Candau, M.D., Director-General, WHO; David B. Allman, President, AMA; William Menninger, M.D., President, Menninger Foundation, Topeka, Kansas.

The opening meeting of the house of delegates will convene Wednesday, May 21, at 2 p.m., a change from the week end opening sessions of the past.

Dr. David B. Allman, President of the American Medical Association. will be speaker at the annual banquet Friday, May 23, at 7 p.m. in the Hall of States, Hotel Leamington. Fifty Club certificates, Southern Minnesota Medical Association Medal, and Distinguished Service Award presentations will be made in conjunction with the banquet.

Open house will be held Thursday, May 22, at 9 p.m. in the Hall of States, Leamington Hotel. Members of the Hennepin County Medical Society will be hosts for the occasion.

Committee breakfasts will be held Thursday and Friday mornings, May 22 and 23, at 8:00 a.m.

The dinner for new members and county officers will be held at 6 p.m., May 21, Town Hall, Hotel Leamington.

The scientfiic program sessions will include panel discussions on the following topics: Emergencies; The Family Doctor's Responsibility to His Pre-School Eye Patient; What's New in Dermatology; Surgery; Viruses and Virus Diseases; Mental Health; Medico-Legal Aspects of Heart Disease.

Lectures to be presented include: Arthur H. Sanford Lecture in Clinical Pathology, Minnesota Division of the American Cancer Society Lecture, Russell D. Carman Memorial Lecture and Northwestern Pediatric Society Lecture.

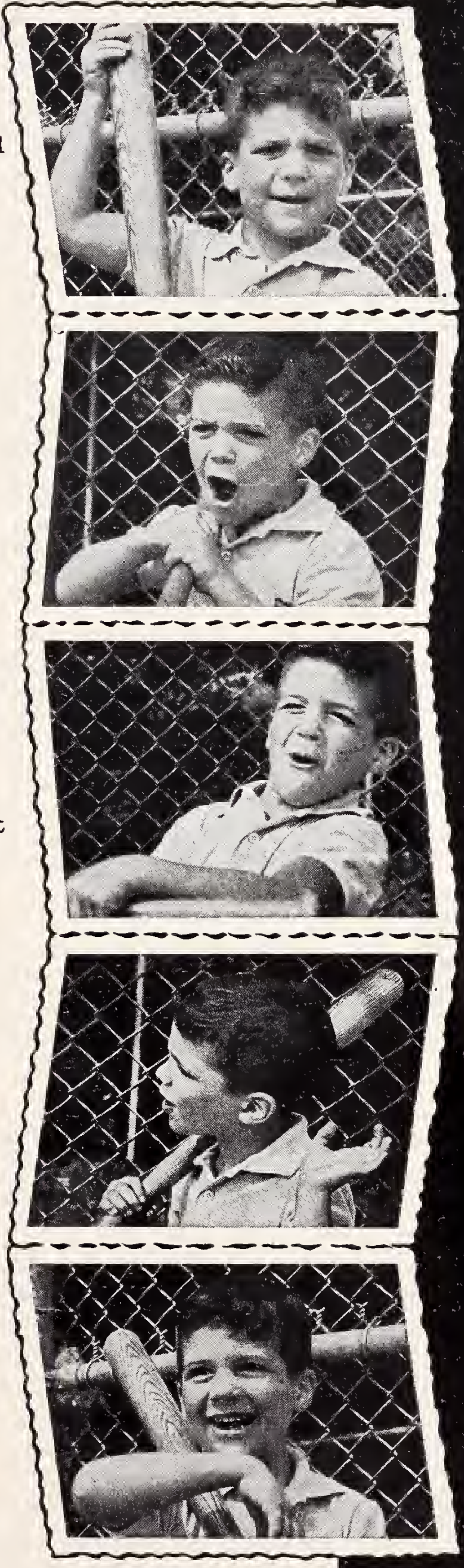
Specialty groups which have accepted dinners or luncheons during the annual meeting include Academy of General Practice, Medical Women's Association, Minnesota Academy of Ophthalmology and Otolaryngology, Radiological Society of North America, and Society of Clinical Pathologists.

For the sportsminded, the annual trap and skeet shoot will be held Saturday, May 24, from 11 a.m. to 4 p.m. at the Twin City Hopkins Gun Club. Dinner, to which wives are invited, will be served at the Edina Country Club at 6:30 p.m.

Golfers will be able to catch up on their favorite

(Continued on Page A-46)

MY DAD— HE HURT HIS BACK REAL BAD



"It happened at work while he was putting oil in something"

"He told Mom his shoulder felt like it was on fire"

"He couldn't swing a bat without hurting"

"But Doctor gave him some nice pills--and the pain went away fast"

"Dad said we'd play ball again tomorrow when he comes home"

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Each PERCODAN* Tablet contains 4.50 mg. dihydrohydroxycodeinone hydrochloride, 0.38 mg. dihydrohydroxycodeinone terephthalate, 0.38 mg. homatropine terephthalate, 224 mg. acetylsalicylic acid, 160 mg. phenacetin, and 32 mg. caffeine.

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STATE MEETING HAS CENTENNIAL FLAVOR

(Continued from Page A-44)

pastime Wednesday, May 21, from 11 a.m. to 1 p.m. at the Edina Country Club. A dinner for physician golfers and their wives will be served at 6:30 p.m.

Official programs will be mailed to all members. Reservation blanks for those activities requiring advance reservation will be enclosed with the program, for the convenience of those planning to attend.

POSTGRADUATE REFRESHER COURSE IN HAWAII

From August 5 to August 21, 1958, the University of Southern California School of Medicine will hold a postgraduate course in Honolulu and on board the S.S. Matsonia. The course will center around actual case histories, which will be used to emphasize diagnostic and therapeutic features.

Price range from $382.15 to $657.25, plus tuition $125.00.

Information concerning this course may be obtained from the Director of the Postgraduate Division, USC School of Medicine, 2025 Zonal Avenue, Los Angeles 33, California.

CERVICAL SMEAR PROGRAM SPEAKERS AVAILABLE

The Professional Education Committee of the American Cancer Society, Minnesota Division, in an effort to co-ordinate the state-wide adoption of Cervical Smear Programs is eager to provide speakers to the various county medical societies or better yet to the group medical societies for the purposes of acquainting the medical group with the most recent information on this procedure as well as the more practical aspects of how the tests should be performed and the follow-up recommendations. Secretaries of those groups interested in speakers should contact Mr. Allan Stone, Executive Secretary, American Cancer Society, 3702 East Lake Street, Minneapolis 6, Minnesota.

AMERICAN HEART ASSOCIATION PAPERS REQUESTED

The American Heart Association will hold its thirty-first annual scientific sessions, October 24-26, 1958, in San Francisco, California.

Applications for presentation of papers at the 1958 Scientific Sessions may be obtained from Dr. F. J. Lewy, Assistant Medical Director, American Heart Association, 44 E. 23rd Street, New York 10, New York. Abstracts must be submitted before June 13, 1958.

PSYCHIATRY FELLOWSHIP TO CONTINUE

The American Psychiatric Association has announced a $100,000 grant from the Smith, Kline & French Foundation to continue the Foundation's fellowships in Psychiatry through 1960.

Seven main types of Smith, Kline & French Foundation Fellowships are available: "A"—support for advanced training of full-time staff psychiatrists of public mental hospitals and schools for the retarded; "B"—awards to hospitals for visiting lectureships and for teaching fellowships; "C"—support to medical schools, teaching centers, et cetera, for extension training programs; "D"—student fellowships to encourage talented medical students to broaden their experience in psychiatric approaches and practice; "E"—medical fellowships to encourage broadened skill in the handling of psychiatric problems by physicians other than psychiatrists; "F"—foreign scholar lectureships to bring outstanding men to the United States; "G"—residency training fellowships under unusual circumstances.

Applications for consideration in May and October must be received by the Fellowship Committee by April 1 and September 15. Information and application forms may be obtained from the Committee, Box 7929, Philadelphia.

CARDIOVASCULAR FELLOWSHIPS AVAILABLE

The Minnesota Heart Association has invited applications for research fellowships and research grants for the fiscal year beginning September 1. All areas of cardiovascular disease study will be considered.

Grants are to be allocated from public contributions to the 1958 Heart Fund drive.

Requests from qualified persons must be submitted to the Association office, 1821 University Avenue, Saint Paul 4, Minnesota, by May 15, 1958. Application forms can be obtained from the Minnesota Heart Association.

POSTGRADUATE COURSES ANNOUNCED

"The Physiological Basis of Clinical Electrocardiography" is offered by the New York University Post-Graduate Medical School during the week of June 2 through 6. Tuition $75.00.

Occupational Medicine, an eight week full-time course for practicing physicians, will be given September 15 through November 7, 1958.

Information may be obtained through the office of the Associate Dean, New York University Postgraduate Medical School, 550 First Avenue, New York 16, N. Y.

PUBLIC HEALTH INFORMATION

Six hundred dailies, weeklies, radio and TV stations in Minnesota now receive the "How's Your Health?" feature each week. The articles compiled by the public relations department of the Minnesota State Medical Association feature timely health topics. All Minnesota newspaper editors regularly receive "How's Your Health?" as a part of their weekly Minnesota Editorial Association mailing. Members are urged to encourage editors who do not use the feature to do so. Similarly, a compliment to editors who publish "How's Your Health?" might be good public relations on the local level.

Ten 20-second sound-on-film health jingles have been placed in the film libraries of the eight Minnesota commercial TV stations. The spots are based on well-known nursery rhymes.

Stations which have received the jingles to be used on a Public Service basis include: WCCO-TV, KSTP-TV, WTCN-TV and KMGM-TV in the Twin Cities; WDSM-TV and KDAL-TV in Duluth; KROC-TV in Rochester and KMMT-TV in Austin.

"Doctor Tell Me," the Minnesota State Medical Association radio health program, is now heard on twenty-two Minnesota radio stations.

If Monilial overgrowth is a factor

Achrostatin* V

TETRACYCLINE (PHOSPHATE-BUFFERED) AND NYSTATIN

Combines Achromycin V with Nystatin

SUPPLIED:

CAPSULES contain 250 mg. tetracycline HCl equivalent (phosphate-buffered) and 250,000 units Nystatin. ORAL SUSPENSION (cherry-mint flavored) Each 5 cc. teaspoonful contains 125 mg. tetracycline HCl equivalent (phosphate-buffered) and 125,000 units Nystatin.

DOSAGE:

Basic oral dosage (6-7 mg. per lb. body weight per day) in the average adult is 4 capsules or 8 tsp. of Achrostatin V per day, equivalent to 1 Gm. of Achromycin V.

Achrostatin V combines Achromycin† V ...the new rapid-acting oral form of Achromycin† Tetracycline...noted for its outstanding effectiveness against more than 50 different infections ...and Nystatin...the antifungal specific. Achrostatin V provides particularly effective therapy for those patients prone to monilial overgrowth during a protracted course of antibiotic treatment.

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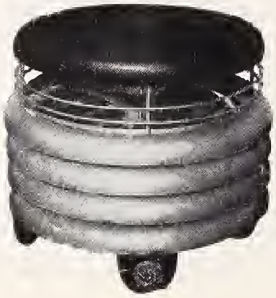

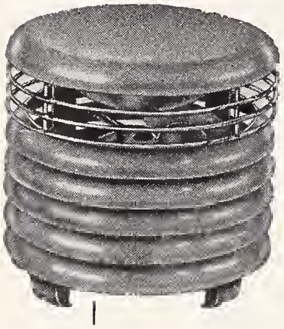

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MM-558a

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Woman's Auxiliary

COUNTY AUXILIARY NEWS

McLeod County

On Wednesday, March 19, the Woman's Auxiliary to the McLeod County Medical Society honored Mrs. C. L. Oppegaard, Crookston, president, and Mrs. Reuben Erickson, Minneapolis, president-elect of the Minnesota Woman's Auxiliary, with a luncheon at Bob's Supper Club in Hutchinson. The occasion celebrated the tenth anniversary of the organization of the McLeod County unit.

Of the thirteen charter members, eight are still active in the present membership of eighteen. The group's first president was Mrs. A. M. Jensen of Brownton. Successive presidents were: Mrs. A. Neumaier, Glencoe; Mrs. D. Brink, Hutchinson; Mrs. J. D. Selmo, Norwood; Mrs. J. J. Smyth, Lester Prairie; Mrs. E. W. Lippmann, Hutchinson; Mrs. K. Peterson, Hutchinson; Mrs. Chas. Sheppard, Hutchinson; Mrs. C. W. Truesdale, Glencoe; and Mrs. M. M. Howell, Glencoe.

Guests included thirty Auxiliary members from seven surrounding counties. Bob's Supper Club had been transformed into a spring garden by the decoration committee, Mrs. Carl Bretzke and Mrs. George Smith of Hutchinson. Tickets chairman was Mrs. A. M. Jensen, Brownton. Mrs. C. W. Truesdale, Glencoe, Mrs. J. J. Smith, Lester Prairie, and Mrs. J. D. Selmo, Norwood, comprised the arrangements committee, and Mrs. Chas. Sheppard, Hutchinson, was the hospitality chairman.

Mrs. M. M. Howell, Glencoe, president, presided. The speakers' table was decorated with old fashioned bouquets of tulips, daffodils and hyacinths, centered with an arrangement of spring flowers in a straw hat. Mrs. Oppegaard addressed the group on "The Role of the Woman's Auxiliary." Mrs. Erickson outlined the plans of the forthcoming WHO convention to be held in Minneapolis in the early summer. The group enjoyed a discourse on "Where Did You Get That Hat?" given by Mrs. E. J. Gavin of Glencoe, highlighted by her display of hats and points on making them.

Following the meeting Mrs. Oppegaard and Mrs. Erickson drove to Redwood Falls to attend a regional meeting of the Auxiliary.

Ramsey County

Instead of the regular February luncheon meeting, the Ramsey County Auxiliary attended area meetings in the homes of members. These coffee parties were arranged by Mrs. Wallace Gleason and Mrs. Malcolm Pearson, co-chairmen.

In March, members were guests of the Auxiliary at a luncheon and style show at the Saint Paul Women's City Club. Mrs. Dorothy Krebs of Frank Murphy's presented a collection of hats which were modeled by members. "Millinery Memories" was the title of the Vivien Grace Gibson collection of antique hats shown by Mrs. Curtiss Oberg. The program was planned by Mrs. John Meade and Mrs. Burtis Mears.

Mrs. Donald Derauf, legislation chairman, prepared a report on pertinent bills in Congress—the Forand and the Jenkins-Keogh bills. Mrs. Rodney Sturley, introduced by Mrs. Herman Wolff, president, presented a paper.

Tribute was paid to the memory of Mrs. A. E. Nichols, who served as president of the Auxiliary during the 1938-39 period.

A letter from a University medical student, thanking the Auxiliary for a $200 scholarship he had received from the Auxiliary, was read.

The Auxiliary assisted Ramsey County Medical Society in publicizing a high school essay contest on "The Advantages of Private Practice in Medicine." Dr. Francis Lynch presented the monetary awards, which had been donated by the Medical Society, to the winners.

Zumbro Valley Auxiliary

The March meeting of the Zumbro Valley Medical Auxiliary was held on Wednesday, March 12, at 2:30 P.M. at the Mayo Foundation House.

Tea was served, followed by a business meeting. A film on Traffic Safety, sponsored by the AAA was then shown.

REMINDERS ABOUT THE 36TH ANNUAL MEETING

Thursday, May 22

Registration—8:30 a.m.
Executive Board Meeting—9:45 a.m.
Executive Board Luncheon—12:15 p.m.
Hennepin County Historical Society Tour—2:30 p.m.
Tea—3:00 p.m.
Open House—9:00 p.m.

Friday, May 23

Registration—8:30 a.m.
Annual Meeting—9:30 a.m.
Annual Luncheon—1:00 p.m.
Post-Convention Board Meeting—3:30 p.m.
Annual Banquet—7:00 p.m.

An official program will be mailed at an early date. In addition, a preliminary program of Woman's Auxiliary activities appears in the April issue of this publication. Remember to make all necessary reservations early. Need more be said?

HEALTH CAREERS FILM PREVIEW

"Helping Hands for Julie" is the title of a new health careers film, co-sponsored by the American Medical Association and the American Hospital Association, under a grant from E. R. Squibb and Sons.

This film is to be completed and ready for previewing during National Hospital Week, May 11 to 17, for the exclusive purpose of advance showings to vocational guidance counselors.

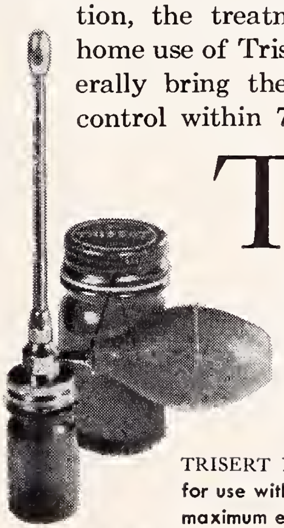
County Auxiliary officers may wish to contact local hospitals to offer co-operation in the sponsorship of this film.

The Woman's Auxiliary is sponsoring a special show-

(Continued on Page A-56)

For fast, complete treatment of vaginal infections

Problem is: she'll wait until discomfort is acute and then expect immediate relief. The answer is Trisert. Trisert preparations contain ALLANTOIN, an effective debriding agent which quickly dissolves heavy mucus often accompanying vaginal infections . . . METHYLBENZETHONIUM CHLORIDE, a quaternary germicide which removes unpleasant odors . . . SUCCINIC ACID, an aid in maintaining optimal vaginal pH . . . 9-AMINOACRIDINE HYDROCHLORIDE which has been included to supplement the bactericidal and trichomonacidal activity of other constituents. Treatment with Trisert Powder will control symptoms fast . . . usually within an hour . . . and provide effective initial treatment for 48 hours. After a second insufflation, the treatment is completed with at home use of Trisert Tablets which will generally bring the infection under complete control within 7 days.



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MM-558b

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General Interest

Dr. Charles C. Cooper, Saint Paul, was named vice president of the American Academy of General Practice at its meeting in Dallas.

* * *

Dr. Harry M. Weber, head of the section of diagnostic roentgenology at the Mayo Clinic, delivered the Fred Jenner Hodges lecture on radiology April 11 at the University of Michigan.

* * *

Dr. Stanley Karansky, a member of the section of anesthesiology of the Mayo Clinic since 1955, has left Rochester for Phoenix, Arizona, where he will carry on a private practice of anesthesiology.

* * *

Two Mayo Clinic staff men were on the speaking program of the sectional meeting of the American College of Surgeons in Salt Lake City. **Dr. John C. Ivins** participated in a symposium on trauma and **Dr. Thomas T. Myers** participated in a panel on peripheral vascular disease.

* * *

Dr. J. Arnold Bargen, chairman of the section of gastroenterology in the Mayo Clinic, and professor of medicine in the Mayo Foundation, has been appointed to the medical advisory board of the National Foundation for Research in Ulcerative Colitis, of Washington, D. C.

* * *

Two University of Minnesota medical professors have won Purdue Frederick medical achievement travel awards to enable them to attend medical meetings of their choice in the United States and Europe.

They are **Dr. C. Walton Lillehei**, professor of surgery in the school of medicine and originator of the world-famed cross-circulation "open heart" surgery technique, and **Dr. Edgar V. Allen**, professor of medicine at the Mayo Foundation graduate school, Rochester, Minnesota, and a specialist in cardiovascular disease.

The awards are presented by the Purdue Frederick Company, pharmaceutical firm, through the International Council for Health and Travel.

* * *

Dr. Harry B. Hall has been elected chief of staff at Fairview Hospital. Other officers for 1958-59 are **Drs. C. Gordon Watson**, chief-elect; **Kristofer Hagen**, secretary, and **Dale H. Correa**, treasurer.

Doctors attending the annual staff meeting heard Dr. Robert Meller, psychiatric committeechairman, report on the first six months operation of the hospital's 34-bed psychiatric ward.

* * *

Dr. Donald T. Cundy has announced the opening of a new office at 697 Marquette Bank Building in Minneapolis.

* * *

A foursome from Rochester recently left for Europe, where they spent two weeks. **Dr. John Kirklin** and **Dr. Robert Patrick** of the Mayo Clinic and their wives spent a week in Germany and a week in England.

The doctors attended a medical meeting in Munich, Germany, where they both presented papers. Dr. Kirklin also gave a paper at Oxford University.

* * *

Dr. C. Walton Lillehei, University of Minnesota, is the 1958 recipient of the Oscar B. Hunter Memorial award of the American Therapeutic society, it was announced.

The award will be presented June 21 at the group's forty-ninth annual meeting in San Francisco. At the presentation, Lillehei will address the society on his work in "open heart" surgery to correct cardiac defects.

* * *

Dr. N. E. A. Leppo of Duluth turned over his practice to Drs. Ed Jorgenson and James Troutman, effective April 1.

* * *

An emotional outlet, as well as food, shelter and clothing, are needed in caring for the aged, **Dr. Robert J. Goldish**, Duluth, said in a talk to the thirty-seventh annual meeting of the Jewish Social Service Agency.

Families cannot adequately handle the needs of these people and the responsibility for caring for the aged must therefore become increasingly a community endeavor, he said. He spoke at a meeting in the Covenant Club.

* * *

Dr. Charles C. Cooper, Saint Paul, president of the Minnesota Academy of General Practice in 1953, has been elected vice president of the American Academy of General Practice.

* * *

Six Mayo Clinic staff men and members of the faculty of the Mayo Foundation participated in continuation courses in internal medicine and surgery at the University of Minnesota.

Dr. J. A. Bargen was on a panel discussing ulcerative colitis. **Dr. Charles H. Slocumb** discussed the current status of treatment of rheumatoid arthritis at the course on internal medicine.

At the surgery course, **Dr. Philip E. Bernatz** discussed treatment of breast cancer. **Dr. F. Henry Ellis** reviewed benign surgical lesions of the esophagus and also participated in a panel discussion. **Dr. Ward S.** Fowler described pulmonary function studies in relationship to resectability of lung and participated in another general discussion. **Dr. John M. Waugh** outlined treatment of diverticulitis and cancer of the pelvic colon.

* * *

Dr. Rodney F. Sturley, Assistant Professor, Department of Gynecology, University of Minnesota, was a recent speaker at a meeting of the Southwestern Minnesota Medical Society held in Worthington. "The Place of Cytology in the Diagnosis of Cancer" was the subject of Dr. Sturley's address.

(Continued on Page A-52)

And it is, oh, such fun!
And I am sure that we shall rue
The time when we are both
too old to play
The game of "Booh"!
—*EUGENE FIELD*

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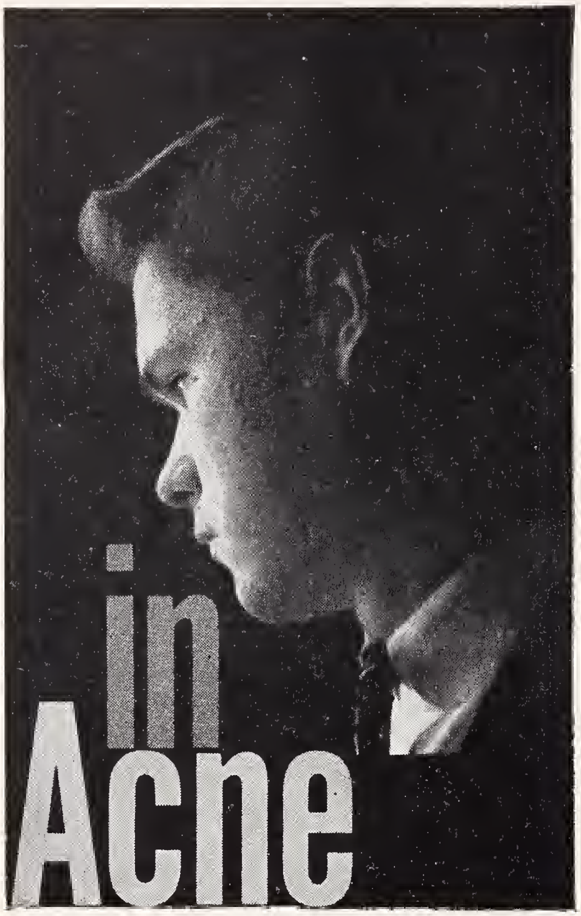

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1. Hodges, F. T.: *GP* 14:86, Nov., 1956.

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(Continued from Page A-50)

Dr. Philip H. Utz, formerly of Rochester and now in private practice at LaCrescent, Minnesota, has joined the staff of St. Francis Hospital at LaCrosse.

* * *

April 27, 1958, was observed as Dr. Patterson's day in Fergus Falls. Residents of Ottertail County and the state joined with Fergus Falls in recognizing the contribution **Dr. W. L. Patterson**, superintendent of the Fergus Falls State Hospital has made to the mental health program of the state. Dr. Patterson has been a member of the hospital staff for forty-six years, serving as superintendent for the greater part of that time.

* * *

Two physicians who have been associated with the Olmsted Medical Group for the past year have elected as partners at the monthly meeting. They are **Dr. Malcolm K. Campbell** and **Dr. William R. Weyhrauch.**

* * *

Dr. Joseph Borg, Saint Paul, has been named to serve on a special advisory committee for Bethesda Hospital. The Committee of prominent Saint Paul men will supervise a drive for funds to finance a 100-bed hospital addition and a 142-bed infirmary across the street from Bethesda. A total of 750 volunteer solicitors participated.

* * *

Dr. Charles W. Mayo has been appointed chairman of a statewide Minnesota World Health Organization and Centennial Committee, it has been announced by Governor Orville Freeman, honorary chairman.

* * *

To express appreciation to **Dr. H. T. Petraborg** who has devoted the past twenty-three years to the people of his community, a project has been started by the Aitkin Community Hospital auxiliary to dedicate the nursery at the hospital in his honor. A nominal contribution of one dollar is being solicited from the 2,500 or more babies delivered by Dr. Petroborg since he started his practice in August, 1935.

* * *

Dr. Frank H. Krusen, head of the Section of Physical Medicine and Rehabilitation of the Mayo Clinic, Rochester, Minnesota, and professor of physical medicine and rehabilitation in the Mayo Foundation, Graduate School, University of Minnesota, has been appointed a member of the National Advisory Council on Vocational Rehabilitation by Secretary of Health, Education and Welfare Marion B. Folsom, Washington, D. C.

* * *

Recent Mayo Clinic Staff announcements include: **Dr. Richard W. Hill**, consultant in internal medicine; **Dr. Robert L. Sommerville**, consultant in medicine; **Dr. Anthony J. Bianco, Jr.**, consultant in orthopedic surgery; **Dr. Horace K. Ivy**, consultant in medicine.

* * *

Dr. Harry M. Weber, head of the Section of Diagnostic Roentgenology of the Mayo Clinic, Rochester, Minnesota, and professor of radiology in the Mayo Foundation. Graduate School, University of Minnesota, gave the

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MIF Infant Food in powder form has been clinically tested by the Department of Pediatrics of a well-known university*, and has been accepted as conforming to the rules of the Council on Foods and Nutrition of the American Medical Association as published in the Journal of the Association page 477, Volume 140, Number 5.
(*Name of university and results of clinical tests sent upon written request.)

APPROXIMATE ANALYSIS

Milk fat	2.60%
Milk protein	2.60%
Milk sugar (lactose)	4.20%
Dextrins, Glucose, Maltose	2.10%
Milk minerals	.62%
Calcium	850 mg. per qt.
Phosphorus	560 mg. per qt.
Iron	5.6 mg. per qt.
Water	88.0%

VITAMIN CONTENT

	Per Qt.
Vitamin A (U.S.P. units)	3000
Vitamin D (U.S.P. units)	800
Vitamin C (ascorbic acid)	100 mg.
Niacinamide	8 mg.
Riboflavin (vitamin B_2)	1.2 mg.
Thiamin (vitamin B_1)	.8 mg.
Pyridoxine (vitamin B_6)	.4 mg.

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INGREDIENTS

annual Fred Jenner Hodges lecture on radiology at the University of Michigan on April 11, 1958.

* * *

Dr. Frederick G. Gunlaugson, Minneapolis, has been named Hennepin County Tuberculosis Control Officer in action taken recently by the Hennepin County Sanatorium Commission. Dr. Gunlaugson currently is director of preventable diseases and tuberculosis control officer for the Minneapolis health department. If appointed to the county office, he would have jurisdiction over tuberculosis matters for the county as well as Minneapolis.

* * *

Dr. William A. O'Brien, Minneapolis, was a guest speaker at the March meeting of the McLeod County Medical Society at Glencoe. His subject was "The Use of Isotopes in the Diagnosis of Thyroid Disease."

Four independent physicians in Hutchinson joined together in group practice as of January 1 and moved into their new quarters, known as the Hutchinson Medical Center, on January 25. Physicians are—**C. O. Bretzke, Kenneth Peterson, George Smith,** of Hutchinson, and **Dan Huebert**, who returned to Hutchinson after completing his general surgical residency at St. Barnabas Hospital in Minneapolis.

* * *

Dr. William S. Eisenstadt, of Minneapolis, spoke before the Blue Earth Valley Medical Society at Fairmont, March 20, on the subject, "The Management of Bronchial Asthma, including Status Asthmaticus."

Doctor Gordon R. Kamman, Saint Paul, and Mr. Charles Murnane, Saint Paul attorney, addressed a joint meeting of physicians and lawyers at Morris, Minnesota, Monday, April 21, 1958. Sixty physicians and lawyers attended from ten surrounding counties. Their topic was "Medico-Legal Problems."

* * *

Only fifteen (0.3 per cent) of 5000 cancers seen in the head and neck service of Memorial Hospital, New York City, were malignant branchiomas. Of these, thirteen men and two women were over fifty years of age. In all these patients, the tumor appeared at about the level of the hyoid bone, the site of the highest nodes in the internal jugular chain, which is also the site in which cervical metastases from a primary in the oral cavity or pharynx are most likely to be found.

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Communication

"McCARTHYISM"

Dear Editor:

I resent the use of editorial pages of Minnesota Medicine to defend recent controversial Supreme Court decisions. I am sure you know it is the opinion of many good legal minds and conservatives in general that the "Warren Court" has run rough shod over judicial precedents, taken to making political decisions, and violated the constitution in case after case.

Doctors have a right to question the motive of anyone using our editorial pages in this manner. However, to quote from a left-wing periodical from socialist England to influence our thinking is absolutely reprehensible. This was done in the January, 1958, Minnesota Medicine under the heading, "The Supreme Court Blow to McCarthyism."

To print something written by someone far removed from the scene, and take no responsibility for the same, is a technique very effectively used by pseudo-liberals as they push us down the road to socialism and the slave state. Please name the person (or persons) on the editorial staff who put this in the journal in order that he may be confronted.

I shall expect an early reply.

Very truly yours,
J. R. Nickerson, M.D.

Fairmont, Minnesota
March 12, 1958

Editor's Note: Doctor Nickerson, your unrepressed anger concerning the editorial on "McCarthyism" is undoubtedly shared by other true, red-blooded Americans of pure, stainless steel quality. You undoubtedly are a man of action and not one to sit back and simply criticize what others do. This is a great opportunity for you to write one or more editorials covering the subjects of "McCarthyism as Viewed by a Conservative," "The Warren Court," "Down the Road to the Slave State," "Our Pseudo-Liberal Editorial Staff" or any other political subject which may come to mind.

The editorials in Minnesota Medicine generally have educational value but are frequently dull and nonstimulating. A contribution from you might start a trend among Minnesota physicians to use their journal to express pent-up feelings on a variety of subjects. Simple "Letters to the Editor" will suffice, but a more polished "Editorial" will be more effective. Incidentally, I shall take no responsibility for the opinions expressed in your editorials and they must be signed by you.

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Book Reviews

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

ULCERATIVE COLITIS. Harry E. Bacon, B.S., M.D., ScD., LL.D., F.A.C.S., F.A.P.S. Professor and Head of Department of Proctology, Temple University Medical Center, Philadelphia; President, American Board of Proctology; Diplomate, American Board of Surgery and American Board of Proctology; Honorary Fellow, Royal Society of Medicine, Philippine College of Surgeons, Brazilian College of Surgeons, Japanese College of Surgeons. Foreword by Alton Ochsner, B.A., M.D., Sc.D., F.A.C.S., F.A.P.S. 395 pages. Illus. Price $15.00, cloth. Philadelphia: J. B. Lippincott Co., 1958.

ANTIBIOTICS ANNUAL, 1957, 1958. Edited by Henry Welch, Ph.D., and Felix Marti-Ibanez, M.D. 1070 pages. Illus. Price $12.00, cloth. New York: Medical Encyclopedia, Inc., 1958.

DIABETES AS A WAY OF LIFE. T. S. Danowski, M.D. Renziehausen Professor of Research Medicine, University of Pittsburgh School of Medicine; Senior Staff Physician at Presbyterian Woman's, Children's, Elizabeth Steel Magee, and Shadyside Hospitals of Pittsburgh; Consultant in Metabolism, Oakland Veteran's Administration Hospital, Pittsburgh. 177 pages. Illus. Price $3.50, cloth. New York: Coward-McCann, Inc., 1957.

OPERATIVE OBSTETRICS. By R. Gordon Douglas, Professor of Obstetrics and Gynecology, Cornell University Medical College, and William B. Stromme, Attending Obstetrician and Gynecologist, Northwestern Hospital and Fairview Hospital, Minneapolis. Foreword by Nicholson J. Eastman. 735 pages. Illus. Price $20.00. New York: Appleton-Century-Crofts, Inc., 1957.

Operative Obstetrics is a welcome addition to obstetrical literature and this volume should be useful to any practitioner specialist and student interested in this specialty.

The authors have organized their material to include all aspects of the practice of obstetrics, and each chapter contains basic practical information. A large number of photographs and diagrams demonstrate the techniques which the authors describe.

In the future, I hope the authors will revise the book as the need arises to keep abreast of modern trends so that this text can be perpetually used as a handy reference for physicians. It should be readily available in the hospital library.

Joseph F. Melancon, M.D.

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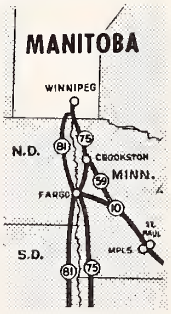

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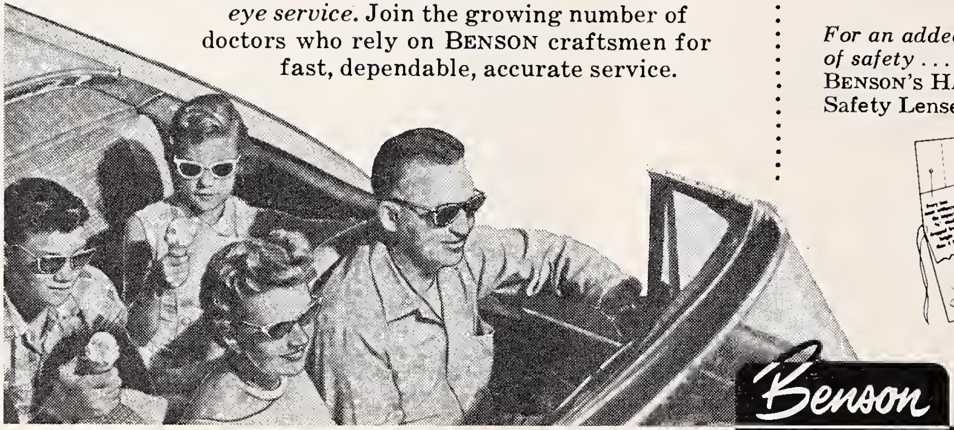

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(Continued from Page A-49)

ing of this film during the annual meeting in San Francisco, Wednesday, June 25, 3:00 P.M., Gold Room, Fairmont Hotel.

The film will be available to state and county medical societies for non-theatrical showings July 1, 1958.

AMA AUXILIARY PLANS JUNE CONVENTION

The call of the West will be heeded by physicians' wives as they travel to San Francisco in June for the thirty-fifth annual convention of the Woman's Auxiliary to the American Medical Association at the Fairmont Hotel. National committee meetings and round-table discussions will be held June 21-23 with formal opening of the convention slated for Tuesday morning, June 24. An interesting and varied program is being arranged by co-chairmen Mrs. Matthew N. Hosmer, San Rafael, and Mrs. Samuel R. Sherman, San Francisco.

Business sessions on Tuesday and Wednesday will be devoted to state and national committee reports and discussions on current projects. Tuesday's luncheon in honor of past presidents will feature guest speaker, Mr. Richard H. McFeeley, principal of George School, Bucks county, Pennsylvania. Speaker at Wednesday's luncheon in honor of the president (Mrs. Paul C. Craig of Pennsylvania) and the president-elect (Mrs. E. Arthur Underwood of Washington) will be Dr. David B. Allman, immediate past president of the AMA. At this session, Mrs. Craig will present the Woman's Auxiliary contribution to the American Medical Education Foundation, and Dr. George F. Lull, AMEF president, will present AMEF awards to the auxiliaries.

Election and installation of national officers will be held Thursday morning with adjournment scheduled for noon.

MINNESOTA MEDICINE

Minnesota State Medical Association

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REFERENCES: (1) Roy, T. E.; Collins, A. M.; Craig, G., & Duncan, I. B. R.: *Canad. M.A.J.* 77:844 (Nov. 1) 1957. (2) Schneierson, S. S.: *J. Mt. Sinai Hosp.* 25:52 (Jan.-Feb.) 1958. (3) Hasenclever, H. F.: *J. Iowa M. Soc.* 47:136, 1957. (4) Rhoads, P. S.: *Postgrad. Med.* 21:563, 1957. (5) Caswell, H. T., and others: *Surg. Gynec. & Obst.* 106:1, 1958. (6) Josephson, J. E., & Butler, R. W.: *Canad. M.A.J.* 77:567 (Sept. 15) 1957. (7) Petersdorf, R. G.; Curtin, J. A., & Bennett, I. L., Jr.: *Arch. Int. Med.* 100:927, 1957. (8) Waisbren, B. A., & Strelitzer, C. L.: *Arch. Int. Med.* 101:397, 1958. (9) Holloway, W. J., & Scott, E. G.: *Delaware M. J.* 29:159, 1957. (10) Murphy, J. J., & Rattner, W. H.: *J.A.M.A.* 166:616 (Feb. 8) 1958. (11) Neter, E., & Hodes, H. L.: *Pediatrics* 20:362, 1957. (12) Woolington, S. S.; Adler, S. J., & Bower, A. G., in Welch, H., & Martí-Ibañez, F.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 365.

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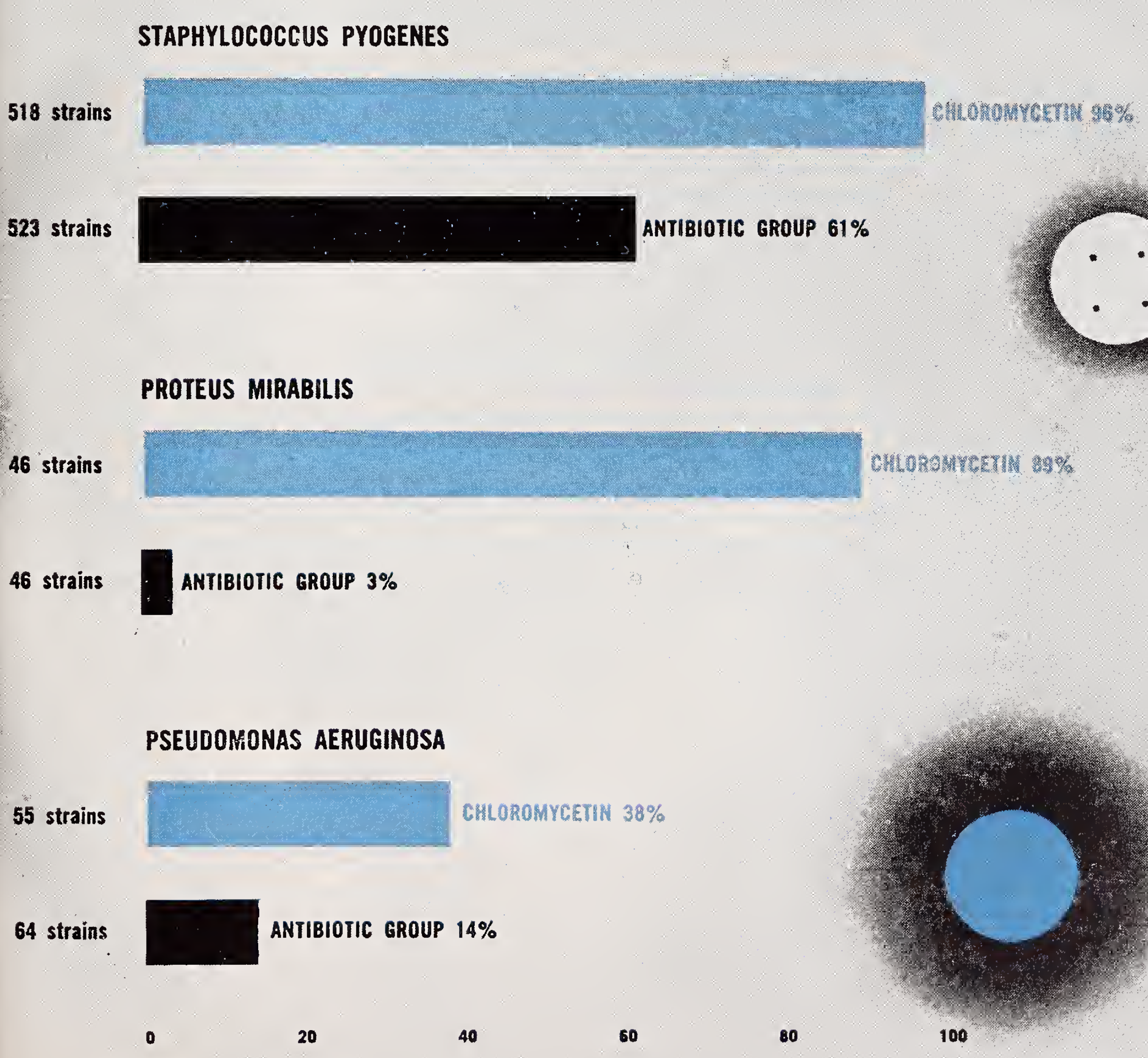

*Adapted from Roy, T. E.; Collins, A. M.; Craig, G., & Duncan, I. B. R.: *Canad. M.A.J.* 77:844 (Nov. 1) 1957.

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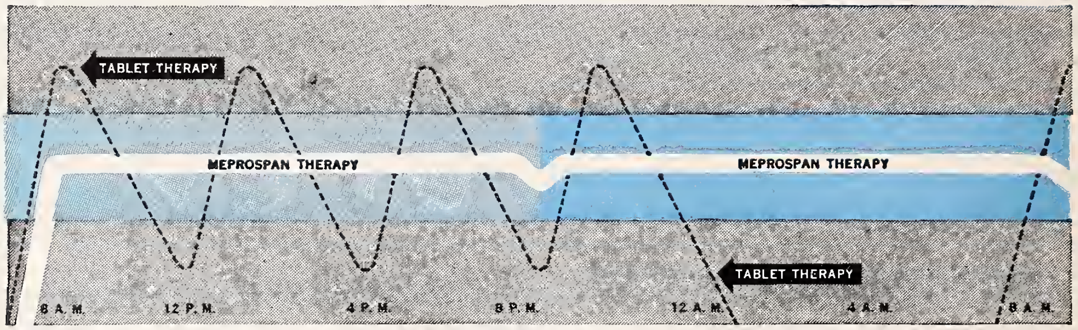

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Original Contributions

Current Appraisal of Cutaneous Drug Eruptions

E. T. CEDER, M.D.
Minneapolis, Minnesota

THE LITERATURE on the subject of drug sensitivity is becoming voluminous and almost every current issue of a medical journal contains a report on an untoward reaction to a drug. By these communications we are informed of their hazards as well as their merits. As is well known, the effects of drugs are not revealed until extensive clinical use has allowed repeated exposure. The incidence of allergic reactions tends to grow as the drug is used more extensively. Formerly drug eruptions were of interest only to the dermatologist and involved only a few drugs such as arsenicals, mercurials, herbs in cathartics, tonics, cough mixtures and a few antiseptics. But medicine is not static and in the past quarter of a century there has been a fantastic increase in the use of hundreds of new chemical agents as diagnostic and therapeutic aids. Not only dermatologists but all physicians in general are now recognizing the increasing incidence of untoward reaction to drugs. Every practitioner of medicine is utilizing a certain number of drugs and it behooves all to be alert and cognizant of their injurious effects. To keep abreast of these developments has become a difficult task as almost daily, hundreds of new drugs are introduced to the medical profession and yes, even to the public.

There are about 150,000 medicinal agents available to the medical profession and that about 10,000 more are being added each year,[1] and since the introduction of the antibiotic preparations about thirty such preparations are in current use, fifteen were introduced in 1955, and five more in 1956.[2] In the past five years a new group of compounds called tranquilizers have appeared and are rapidly increasing in number. There is no doubt that modern drugs are a triumph over disease and have saved millions of lives, ameliorated much suffering, have reduced the existence of many communicable diseases to a minimum and may even in the future eliminate their existence. But with this great evolution in therapy have arisen problems of their hazards.

For the most part, although we have called attention to the several hundred thousand medicaments available, only about a thousand of these are popularly used since a great majority of others are obsolete. Although it is possible in therapy, for any drug to produce a toxic reaction, particularly in a patient of sensitivity, only a few hundred have a more marked capacity to do so. Among the mild or grave reactions, those of the skin are in all likelihood the most common. These also vary in degree of this capacity. Table I illustrates the variation.

TABLE I. COMPARISON OF SENSITIZING PROPERTIES OF DRUGS

Relatively Innocuous	Moderate Sensitizers	Potent Sensitizers
Digitalis	Tranquilizers	Sulfonamides
Vitamins	Tetracyline	Penicillin
Opiates	Bromides	Quinine
Demerol	Resorcin	Butazolidin
Anti-histaminics	Arsenicals	Gold Salts
Choral Hydrates	Phenolphthalein	Mercurials
Procaine	Thiouracil	Barbiturates
I. N. H.	Benzocaine	
	Salicylates	

The cutaneous reactions are essentially of three patterns: eczematous, exanthematoid and urticarial. There are, however, a few other bizarre patterns such as fixed drug, erythema multiforme and "Id" patterns.

As a rule, a specific drug usually produces a distinct pattern, but some drugs have multiple patterns. The fact that certain drugs produce a certain cutaneous eruption enables the physician to suspect a particular drug out of many which the patient may have received. Drug eruptions

---

Inaugural thesis presented before the Minnesota Academy of Medicine, November 13, 1957.

technically include both dermatitis venenata and dermatitis medicamentosa. The former results from direct contact by the offending agent with the skin and is an epidermal sensitivity and dermatitis medicamentosa refers to dermal sensitivity in which the drug was transported systematically. The types of reactions most commonly produced by various drugs are illustrated in the following tables.

It must be borne in mind, however, that any type of cutaneous reaction can be produced by almost any drug and the skin reaction may or may not be accompanied by systemic manifestations such as fever, lymphadenopathy or hemopoietic disturbances.

TABLE II. TYPES OF SKIN REACTIONS

Eczematous Eruption	Scarletinaform and Marbilliform	Erythema Multiforme
1. Sulfonamides	1. Barbiturates	1. Barbiturates
2. Penicillin	2. Sulfonamides	2. Sulfonamides
3. Furacin	3. Tranquilizing drugs	3. Penicillin
4. Mercurials	4. Arsenicals	4. Butazolidin
5. Benzocaine	5. Penicillin	5. Gold Salts
6. Anti-histaminic oint.	6. Quinine	
7. Streptomycin oint.	7. Gold Salts	

TABLE III. TYPES OF SENSITIVITY

Urticarial	Fixed Drug	Furunculoid and Aceneform
1. Penicillin	1. Phenolphthalein	1. Bromides
2. Salicylates	2. Sulfonamides	2. Iodides
3. Carticotropin prep.	3. Aureomycin	
4. Insulin	4. Dilantin	

TABLE IV. SYSTEMIC REACTIONS

Photosensitizing	Exfoliative Dermatitis	Purpuric and Hemotoxic
1. Thorazine	1. Arsenicals	1. Quinidine
2. Sulfanilimide	2. Sulfonamides	2. Sulfonamides (Gantrisin)
	3. Barbiturates	3. Butazolidin
	4. Penicillin	4. Thiouracil
		5. Barbiturates
		6. Salicylates

The commonly used drugs such as penicillin, barbiturates, sulfonamides and tetracyeline group well illustrates these overlapping multiple cutaneous manifestations.

The cutaneous drug eruptions may mimic and simulate many of the dermatological entities and a dermatologist usually bears this in mind constantly in considering the etiology of any dermatosis. The tendency of particular drugs to produce characteristic if not pathognomonic skin response naturally forms an important diagnostic criterion for identification and elimination of offending drugs.

Mechanism of Drug Eruptions

The mechanism for the untoward reactions to drugs has been recently well reviewed by Rostenberg.[3] He relates that the state of hypersensitivity is mediated by several mechanisms consisting of allergy which is an antigen-antibody response, ecologic mechanism, Schwartzman phenomenon and disturbances of the enzymatic systems of the skin. In most instances it is caused by an allergy which is achieved after a period of incubation. This period may range from days to years depending on the susceptibility of the individual—a chance trick of nature which may set up the antigen-antibody complex in a given individual. The state of sensitization depends usually on the chemical nature of a drug, dose and route of administration and frequency of exposure. It has been quite well established by Landsteiner and other workers that the drug is made antigenic by conjugating with body proteins forming a specific chemical proteinate.[4] Thus a specific antibody may be produced. Once acquired, the state of sensitivity may be permanent or transient. Circulatory reagins to some laboratory-prepared drug protein conjugates have been demonstrated in animals.[4] For the most part, the synthesis of these drug proteinates is too difficult to be accomplished in the laboratory. Circulating reagins to the drug or chemical *per se* is not demonstrable and there-

TABLE V. COMPARISON OF PENICILLIN AND SULFONAMIDES

Penicillin	Sulfonamides
1. Urticaria	1. Scarletinaform and marbilliform
2. Bullous	2. Erythema multiforme
3. Flare up of latent fungus infection	3. Urticarial
4. Morbilliform	4. Erythema nodosum (sulfathiozole)
5. Exfoliative dermatitis	5. Purpura (gantrisin)
6. Black Tongue	6. Photosensitization (sulfanilamides)

TABLE VI. COMPARISON OF BARBITURATE AND AUREOMYCIN

Barbiturate	Aureomycin
1. Scarletinaform and morbilliform	1. Pruritus ani et vulva
2. Erythema multiforme	2. Stomatitis
3. Urticarial	3. Black tongue
4. Purpura	4. Fixed drug
5. Pemphigoid	5. Eczematous
6. Fixed drug	

fore, assessment of the state of sensitization is not possible by skin tests with the exception of epidermal sensitivity with a contact patch test. Damashek feels that one can frequently predict the cutaneous and hemotoxic potential of a drug by its chemical configuration. He feels that it is highly possible that the greatest degree of toxicity and of reactivity occur with those drugs which have the largest number of NH_2 and chlorine radicals with a benzene structure.

The purpuras are believed to be due to a platelet antibody and this has been demonstrated in a thrombocytopenic purpura produced by Sedormid and studies by Damashek have demonstrated that in quinidine thrombo-penia, quinidine attaches itself firmly to the platelet and acts as an autoantigen.

The mechanism of some types of cutaneous reactions is not too well understood but the age of chemotherapy is only in its beginning. We can anticipate an increasing demand for clarification of the mechanisms, which underlie such reactions. Mechanisms now explained by analogy with immunologic principles for the most part were established for substances other than drugs and almost exclusively in animals. Because much of the preliminary data regarding toxicity of drugs is obtained in animals and because allergic reactions to any new drug is not common to animals, the capacity of any new drug to produce allergic disease in man must await extensive clinical use allowing repeated exposure as well.[5] An allergic state requires previous exposure to the drug some time previously and presupposes an interaction between a drug and an antibody formed previously. Prolonged exposure, repeated exposure and size of the dosage are factors which predispose to allergic reactions. Oral administration is believed to be less conducive to production of antibodies. Parenterally administered substances are more likely to do so as this route allows the chemical to reach the site of synthesis of the antibody (believed to be a protein and probably a globulin) more expediently and directly.[3] It is believed that slowly absorbed preparations administered parenterally cause greater reactions.

Increasing Incidence of Drug Sensitivity

It has been emphasized that a few drugs have accounted for the greatest incidence of drug reaction chiefly because of their extensive use in therapy. Probably most notable of these is penicillin. About 360 tons of this compound are manufactured annually, constituting 150 million courses of three million units each. It is estimated that the incidence of sensitivity to penicillin is now about 10 per cent of the population. The public is not only exposed to pencillin in medications where it is incorporated in nose drops, throat sprays, troches, chewing gum, besides oral and parenteral administration; furthermore, its use is becoming more common in animal husbandry and small amounts may be found in meat, eggs, milk and cheese. Production figures indicate that few of the populace have not been treated by this drug in the past several years. This constant exposure and re-exposure has become of great concern because of the increased incidence of immediate and delayed reactions.

Corr, in reviewing the subject, compares the annual incidence of severe reaction in the past ten years and shows a geometric increase of anaphylactic reactions in the past five years and the ascending increased rate will probably be projected into the future as the state of sensitization has been set up. About 250 deaths to penicillin reactions were reported in the literature in 1954, but it is natural to assume that these are but a fraction of the actual number occurring throughout the nation. I know of fatal cases and nonfatal cases which have not been reported from this community in the past year. A canvas of 1000 physicians in California disclosed seven deaths in 1954, and since this represents about 0.6 per cent of the physicians in the country, a figure of 1000 fatalities could be estimated in the U. S. at that time.[6] The incidence is undoubtedly higher in 1956, as the curve of expectancy is projected from Corr's figures. This does not include hundreds of near-deaths by anaphylactic shock which are more numerous. It is stated that only 30 per cent of anaphylactic reactions are fatal. The anaphylactic reaction can be provoked by injection, oral, aerosol spray, or topical application. A reaction occurred in a few minutes after insertion of a penicillin lozenge in the mouth[7] and several deaths and other severe reactions have occurred from injection of minute amounts intradermally in the performance of skin tests.[8] A large percentage of serious reactions occurred in allergic individuals and it is stated that 15 per cent of the populace are of atopic or allergic constitution.[9]

It has been speculated that the procaine radical augments the reactivity and that its combination with penicillin promotes a more antigenic compound. Procaine itself, however, has been exonerated as a factor in these reactions as the reactors are not allergic to it *per se*. Procaine penicillin, however, is not alone in producing reactions as many reactions have occurred with other penicillin salts and preparations as well.[7] Recently, oral administration of penicillin has become more widely used, particularly since introduction of Penicillin "V." According to the most recent reports of Magazini, thirteen anaphylactic reactions have been reported by oral administration and he reports one to Penicillin "V." Corr stressed that the dangers of oral use of penicillin has not been sufficiently emphasized and that its widespread use in minimal indications may sensitize an individual to subsequent parenteral administration.[5]

The most common reaction to penicillin is the delayed reaction which is characterized by development of urticaria and augioneurotic edema associated with or without lymphadenitis, fever, and joint pains. This develops in about five to fourteen days after administration, usually at about ten days. This is similar to the pattern produced by foreign serum reactions and it is, therefore, called "serum sickness" type of reaction. Its incidence ranges from 1 to 3 per cent[9] of all cases receiving penicillin. It is self-limiting and gradually abates in from one week to several months.

Any reaction to penicillin can occur even though well tolerated previously. Once an indication of sensitivity has appeared there is no way of knowing whether a patient who has had an allergic reaction to a drug will react again on re-administration. Skin tests are not reliable to determine sensitivity as many false negatives occur.[10] It is, therefore, best to assume that subsequent injections will produce further and even more disastrous reactions. Pillsbury noted an incidence of urticaria of 1.8 per cent in the 824 cases of initial treatment and 6 per cent in 200 cases of retreatment.[11]

Antibiotics in Vaccine Poses Problem

Some concern has been raised on the safety of poliomyelitis vaccine and its hazards of administration to allergic or even non-allergic individuals since it contains 200 units per cc. of penicillin. Reports revealed, however, that rarely has a patient had an allergic reaction after its administration and this is estimated to be about one in a million. Even these could have been coincidental. Many lots of the poliomyelitis vaccine contain no penicillin and one pharmaceutical manufacturer has prepared the vaccine with polymixin. However, the greatest use of these preparations up to now have been in children and the probability of sensitization by previous penicillin is not as great in this group as in the adult population.

Sensitivity to Tranquilizing Drugs

The next most commonly used and prescribed drugs are popularly known as tranquilizing or ataractic drugs. In the past four years, several such preparations have been introduced and reports of their use is not sufficient to quote the incidence of untoward reaction. Chlorpromazine (Thorazine) was the first to be employed and to date some of the untoward reactions or complications that have been reported are obstructive jaundice, agranulocytosis, orthostatic hypotension, toxic delirium and numerous skin reactions.[13] The manufacturer's brochure indicates that the incidence of cutaneous manifestations is about 6 to 9 per cent consisting of morbilliform erythematous eruption, photosensitization (increased sensitivity to sun light), urticarial, petechial and bullous lesions. These are developed with high dosage of 400 mg. or more per day. Most of the lesions disappeared when the dosage was decreased or the drug was discontinued. It appears that there is a critical dosage to produce a reaction and these occur usually only in institutional cases. It is reported that in many instances the reactions are so mild that the therapy was not discontinued and in time spontaneously abated even though the therapy was continued. The exception to this situation was those manifesting photosensitization which persisted at any dosage.

Mephobromate (Equanil) was introduced in July, 1955, and has become widely prescribed. Prescription pharmacists have informed me that one out of every five prescriptions are for one of the tranquilizing drugs and that Equanil or Miltown constitutes the greatest percentage of those. The manufacturer states that over one billion tablets were dispensed in 1956. In spite of this extensive use, we have meager reports of adverse reactions, but this is probably due to the fact that sufficient time has not elapsed for reports of studies to appear in the literature. To date, the reactions affect the cutaneous, muscular, gastro-intestinal and cerebral systems. The cutaneous effects are low in incidence and are characterized by a dense fine erythematous papulo-vesicular eruption over the pelvic girdle, breasts and forearms. Some have intense pruritus with minimal lesions. These reactions occur on the usual dosage of 400 mg. three times daily and promptly disappear on the discontinuance of the drug.[14]

Resurgence of the Clinical Use of Sulfonamides

The sulfonamides are potent sensitizers and the introduction of penicillin was a welcome development. With the troublesome problem of hypersensitivity, drug resistance and superimposed infections encountered with the antibiotics, interest in the sulfonamides is being resumed.[15] In the period following introduction and wide use of penicillin several new sulfonamides are being synthesized. Interest in triple sulfonamides consisting of sulfadiazine, sulfamerazine and sulfamethazine is still maintained and used without as much anxiety as formerly. Lepper found that of forty-eight patients previously sensitive to sulfonamides, 30 per cent failed to develop reactions on a second course and none had anaphylactic reactions.[16]

Treatment of Drug Eruption

Of primary interest is the management of penicillin reactions both of the immediate and delayed types. One is admonished to always inquire as to previous reactions to this drug before administration. If any question exists of such an experience, other antibiotics or chemotherapy should be utilized. When penicillin is injected, it should be made low enough on the lateral aspect of the arm so that a tourniquet can be applied if a reaction develops. The patient should be kept under observation for at least twenty minutes after injection as anaphylaxis can develop during that time. Every physician should be prepared to treat an anaphylactic shock and have epinephrine readily available. If the reaction develops, about 0.5 cc. of epinephrine 1:1000 should be injected at site of the penicillin injection or intramuscularly elsewhere and repeated in five minutes if needed. This may be supplemented by intravenous administration of aminophylline (3¾ grs. per cc.) for bronchospasm and Levophed to combat circulatory collapse. An open air-way should be established and oxygen therapy instituted. Intravenous administration of hydrocortisone can be often life-saving.

In the delayed reactions, characterized by pronounced urticaria, angioneurotic edema, arthralgia and fever can usually only be relieved by the cortisone or one of its derivatives. The use of antihistaminics is usually disappointing and does not bring about a complete and prompt relief of the distressing, incapacitating or dangerous initial phase.[16]

Desensitization to penicillin was accomplished by Alexander[17] in eight cases. He started the procedure with dosage of 250 units given intramuscularly four times daily. The individual dose for each successive day was doubled until 300,000 units per cc. was given. The state of desensitization was known to persist in two of these patients as larger doses of penicillin were administered for subsequent infections at a later date.[17] The value of attempting desensitization is questioned by some. It is a very tedious procedure. Keeney[18] suggests that serious reactions could be avoided by preliminary or concomitant injections of corticosteroids. This was borne out by studies in experimental animals.

The corticosteroids have proven valuable in suppressing severe reactions of all types. The causative agent can usually be identified and withdrawn and only a short period of administration is necessary. Cortisone or its derivatives are preferred to corticotropin, since the former is more rapid in its effect. The corticosteriods probably have their effect on the small arterioles and decrease their permeability.[19] Whether or not the corticosteroids have a capacity to suppress or diminish antibodies in an allergic situation is a moot question.

Caution in the use of antibiotics is advised in many editorials[7,20-21] because of the rapid increase in drug reactions. These emphasize that antibiotics should not be used unless absolutely essential and not for trivial infections, such as the common cold, et cetera. Some authorities are opposed to the use of chemoprophylaxis in "clean" surgery. Despite the popularity of this procedure its value has been considered negligible in few controlled studies.[20]

Since the Federal Food and Drug Administration was created to inform and protect the public, it is important that the labels of drugs and manufacturers' literature carry appropriate contra-indications and warnings of adverse effects of a given product. We need to know as quickly as possible all about a drug reaction and a physician should not have to receive his warnings from published papers which may take many months or few years to appear.

The Council of Pharmacy and Chemistry of the American Medical Association is endeavoring to devise a program in which it can serve as a central reporting agency for adverse reactions to drugs and disseminate this information.[22] Because of the ingenuity and competitive enterprise of pharmaceutical chemists, and mother earth seeming to have a wealth of antibiotic material, I am sure in the next decade we can anticipate a continuous deluge of new medicinal agents. Some problems of untoward drug reactions will be solved by discovery of newer, less toxic preparations. If pharmacological history repeats itself, however, continual usage of new drugs will create new problems of sensitivity.

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INJURIES IN THE UNITED STATES

During the last six months of 1957 about 25 million Americans were injured enough to require medical attention or to limit their activities for at least a day.

Injuries during this period resulted in almost 214 million days of restricted activity, including 55.5 million days spent in bed at home or in a hospital.

These figures were disclosed in a report by the Public Health Service's U. S. National Health Survey. The report also shows:

Home accidents injured 10,065,000 people—or 40.3 per cent of all those injured.

Work accidents injured 4,173,000—or 16.7 per cent.

Motor vehicle accidents injured 2,444,000—or 9.8 per cent.

Other kinds of accidents and injuries resulting from violence involved 8,267,000—or 33.1 per cent.

For this period there were, on an average, about 1,175,000 persons every day whose activities were limited because of injuries. Of these, 305,000 were in bed or in a hospital each day.

Of the total injured, 14.1 million were males and 10.8 million were females; 14.9 million were urban residents; 7.1 million were residents of rural-nonfarm areas; and 3 million lived on farms.

The report is the third in a series based on continuing nationwide household interviews, conducted for the Public Health Service by the U. S. Bureau of the Census, with a representative sample of the population. The information recorded about individuals is confidential, and only statistical totals are published.

The figures are preliminary since they are used on only six months of data collection. They are subject to possible revision when statistics have been gathered for a longer period of time.

The Use of Radioactive Iodine in the Diagnosis and Treatment of Hyperthyroidism

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WILL the use of radioiodine completely displace thyroidectomy as the treatment for hyperthyroidism? More and more reports appear in the literature dealing with this type of treatment, and one might believe that the thyroid surgeon will soon be obsolete. The popularity of the use of I^{131} is increasing and there is no doubt that fewer thyroidectomies are being performed at present. There is a natural tendency for any new addition to our medical armamentarium to be glorified beyond its actual value. The overuse of vagotomy in the treatment of peptic ulcer was a good example of this. I do not believe that I^{131} will be used indiscriminately, but there will be a demand for its use that cannot be substantiated by its actual value as a "cure-all" for hyperthyroidism.

Although we have hardly scratched the surface of a great new field of medical enterprise, our present knowledge of the use of radioactive material indicates that progress will be made within this decade.

Inasmuch as the thyroid gland is iodophilic and does not discriminate between an atom of iodine that is radioactive and one that is not, our greatest use of radioactive iodine is in the diagnosis and treatment of thyroid disorder. Hamilton, in 1942, used an iodine isotope in the treatment of an overactive thyroid. Others followed his idea, but, aside from the original literature concerning his work, little was published until 1947. This lapse paralleled the period of secrecy during which the atomic bomb was developed. After World War II the huge neutron source of the Oak Ridge reactor began to produce isotopes that could be used in industry and medicine. With the production of I^{131} (half-life of eight days) it was now possible to supply medical centers throughout the country with a new agent for the diagnosis and treatment of hyperthyroidism.

Recently, there has been a great increase in the number of papers appearing in our medical literature relating to the use of radioactive iodine. Inasmuch as I was associated with the development and production of atomic energy during the war, one might assume that my remarks would be those of an isotopist. However, I am writing this as a clinician and shall present my ideas accordingly. The thesis is based on the observation of over 2,000 patients who were examined at Saint Joseph's hospital over a period of four years. Of this group I shall discuss only the hyperthyroid patients who were treated with I^{131}.

The diagnosis of hyperthyroidism is not always an obvious one. Each individual suspected of having an overactive thyroid gland with an associated excess production of thyroxine, should be examined by using all of the available tests if necessary. If we find a patient whose symptoms are so outstanding that we are quickly able to conclude that his thyroid is putting out too much thyroxine, the BMR or the iodine uptake alone may be sufficient. On the other hand if only a few clinical symptoms such as tremor, fatigue, and weight loss are present, we cannot depend on either of these tests alone.

The PBI^{131} conversion ratio is a valuable test of thyroid function and is the one single procedure that we consider most valuable. By this procedure the radioactivity of plasma containing protein bound iodine is compared to that of plasma containing free iodine. Separation of the two fractions of plasma is accomplished by passage through a column of resin which acts as an ion exchange agent. We have compared the latter procedure with that in which separation is obtained by precipitating the protein-bound iodine fraction from plasma using trichloracetic acid as a precipitant. The former method is a more practical one and we have found it to produce accurate results. The PBI^{131} conversion ratio enables the clinician to distinguish clearly between borderline hyperthy-

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roidism and the euthyroid state. The normal range is from 5 per cent to 50 per cent.

There are two pathways open to iodine when it gets into the blood stream: it may be trapped by the thyroid gland, or it may be excreted by the kidney. The portion not trapped will be excreted in the urine. By determining the activity of radioiodine in a twenty-four hour specimen of urine, we have a check on the measure of activity found in the thyroid. It is ordinarily possible to account for at least 80 per cent of the I^{131} ingested by the patient. This test serves its greatest purpose when the results are unusual, that is, if the uptake is low and the urinary activity is low. Such findings indicate the possibility that thyroid tissue is present in other than the cervical region. Metastasis of thyroid cells to bone often produces this picture.

It is possible to get some idea of thyroid function by determining the amount of radioiodine activity in the patient's saliva. Jaimet and Thode have used this method for determining hypothyroid states in borderline cases. They have found that this activity is high when the PBI conversion ratio is low. The opposite results when the thyroid gland is overactive. In our laboratory we do this test routinely on all patients and have found that it serves as a valuable adjunctive agent. At the present time our results on the tests of the 2,000 patients are being reviewed. This will be covered in a subsequent paper.

In order of value as diagnostic procedures, we consider the following important in determining the activity of the thyroid gland: (1) PBI^{131} conversion ratio, (2) uptake studies, (3) saliva activity, and (4) urine activity. Of course we are not excluding chemical determination of PBI. This, if performed accurately, is of tremendous value and ranks with the PBI^{131} conversion ratio. In this paper I am including only radioactivity tests. All of these tests are of no value unless they conform to some degree with clinical findings.

Before we treat a hyperthyroid individual with radioiodine, there are several problems that we must consider. The age of the patient is an important factor. It is now generally believed that only those over forty to forty-five years can be safely treated. Although it has never been proved that cancer of the thyroid develops after the use of radioiodine, the possibility of such should be considered as a potential danger. It is wiser, therefore, to avoid the risk involved in treating younger patients unless no other treatment is available or advisable.

The question of whether or not multiple adenomatous or nodular goiters should be treated with radioiodine is considered by some to be an important one. My principle is: to treat only those, such as the thyrocardiac individuals, who are too ill to undergo surgery. Although we scan the nodular glands and are unable to find "cold nodules" even with the scintogram, it is not always possible to rule out malignant nodules by this method. Therefore I believe that thyroidectomy is the treatment of choice. If there is a persistence or recurrence of overactivity following surgery, we then treat the patient with radioiodine.

The principles of treating adenomatous goiter do not generally apply to diffuse hyperplastic glands. I believe that all patients over the age of forty to forty-five who have the latter type of hyperthyroidism can be treated with radioiodine. Many considerations enter into the administration of radioiodine to patients with Graves disease, and, to some degree, greater care in evaluating the individual to be treated is necessary. I would no more give an average dose of I^{131} to an acutely toxic individual than I would advise surgery before the proper preoperative program has been carried out. After the ingestion of radioiodine, moderate edema occurs within the thyroid and a washing out process takes place which throws an excessive load of preformed thyroxine into the blood stream. There is then the possibility of thyroid crisis with all the attendant dangers that occur with thyroid storm. To offset this we give propylthiouracil for several days before treatment. In this way the thyroxine is held within the gland. The anti-thyroid drugs do not interfere with the pick-up of I^{131} from the blood stream and do not affect the amount of internal radiation within the gland.

There are two factors that enter into the determination of the total dosage of radioiodine. These are the size of the gland and the effective half-life of I^{131} within the gland. I have yet to hear two physicians agree on the size of any single thyroid gland. At best, the calculated size is almost the product of guess work. It is somewhat like the man who had no scales with which to weigh a hog he wished to sell. He used the ordinary teeter-totter by tying the hog to one end and piled rocks on the other until a balance was reached. He then guessed at the weight of the rocks. In our laboratory I judge the size of the gland as one plus, two plus, three plus, and four plus. One and two plus is anything under thirty grams, and two and four plus is anything above thirty grams.

The effective half-life is determined by taking uptakes at twenty-four, forty-eight, and seventy-two hours. The half-life is reached when the percentage of uptake is one-half that of the original uptake. The shorter the half-life, the more rapidly iodine leaves the gland and less radiation effect results. The reverse is true if the half-life is longer. Therefore, a thyroid gland demonstrating a short effective half-life of I^{131} requires a larger dose for proper radiation than for one with a longer half-life. We do not treat any patient without having this information beforehand.

I prefer the single dose treatment if possible. However, there is less possibility of producing hypothyroidism by dividing the dosage into two or three and giving them at intervals of three to four months. In the early days when we treated younger patients, we were more likely to produce hypothyroidism by the single dose method. All of our patients are asked to return three months after the original treatment. The clinical appearance and the BMR will tell us whether or not another uptake study should be done. If after this second test we believe that there is too much overactivity of the thyroid gland, we give approximately one-half of the original dose of I^{131}. During the past two years I have had the experience of examining a patient who, at the end of three months, indicated thyroid activity which ordinarily would be treated again. However, I did not treat him but waited another three months. I was then surprised to find that a euthyroid condition was present. Since then I have been less anxious to administer radioiodine a second time unless there was definite evidence that the full effect of the original dose had been reached. Prudent clinical judgment is necessary to make this decision.

I have been asked frequently about the possibility of uncomfortable side effects which are associated with the use of radioiodine. Some patients complain of unusual weakness and palpitation which may last for several days. This is more likely to occur in those who have received a full dose of I^{131}. These side effects may result from the extrusion of a large amount of thyroxine from the gland. As I mentioned earlier, the administering of an anti-thyroid drug before treatment will prevent much of this. Unfortunately, it is difficult to predict the action of a gland before treatment, and we may overlook the possibility of an overload of thyroxine after the treatment. As one gains experience in handling hyperthyroid patients, the pre-treatment evaluation will improve.

There are two minor symptoms that deserve mention. Mild thyroiditis, which is manifested by tenderness of the gland, occurs frequently. It usually disappears within two weeks. A burning of the tongue is often manifested two or three days after the treatment, but it disappears quickly. We must remember that there is some radioactivity of the saliva which lasts from twenty-four to forty-eight hours after the ingestion of I^{131}. It is also possible that a low grade glossitis might occur.

Does radioiodine increase the degree of exophthalmos? From the experience obtained in our laboratory I would say that it occurs less frequently in these cases than in those who have been surgically treated. Dobyns and Haines found that two-thirds of a surgically treated group showed an increase in exophthalmos. In our small series only 20 per cent of exophthalmic patients treated with radioiodine became obviously worse. Forty per cent remained unchanged while forty per cent improved. These figures have been substantiated by others who have treated larger groups.

Summary

In summary, we say that the use of radioiodine in the diagnosis and treatment of hyperthyroidism is increasing in popularity. No diagnostic agent is of real value unless used as an adjunct to the clinical picture, and there are no hyperthyroid individuals devoid of clinical symptoms. The uptake study is of little value if used alone. The PBI^{131} conversion factor is a test of great importance. Determination of saliva activity is a valuable check on the PBI^{131} conversion ratio just as the determination of urine activity is a check on the uptake of radioiodine.

We do not treat patients unless they are at least forty years of age. This principle, although based on theory alone, should be followed until more substantial information is obtained. We treat toxic adenomatous or nodular goiters only if surgical treatment is not practical. On the other hand we treat all cases of Grave's disease unless age contraindicates.

The minor effects of internal radiation have been discussed. The possibility of aggravating exophthalmos by using radioiodine seems to be less than by surgical treatment.

We have purposely avoided discussing this thesis in a detailed manner and have avoided technical details so as to present our ideas in a general way. This work is based on the study of 2,000 patients who were examined in our laboratory at Saint Joseph's hospital.

In conclusion I may say that the use of radioiodine should be directed by one who is trained in clinical medicine as well as in the use of isotopes regardless of his specialty. I may also predict that the thyroid surgeon will not become obsolete in the near future!

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MENTAL ILLNESS IS LARGE HEALTH PROBLEM

The mental hospital population in the U. S. has declined for the second successive year but admissions continue to climb. Graphic evidence of these trends is presented in a recent issue of *Patterns of Disease.*

The publication reports that new forms of treatment for the mentally ill are shrinking their periods of hospitalization.

The downward trend began in 1956 with about 8,000 fewer persons in the public mental hospital population than in 1955. However, there were about 3,000 more first admissions in 1956 than in 1955. *Patterns* points out that studies are needed to determine whether this is due to an increase in mental illness itself or "merely an increased use of expanding facilities."

The new tranquilizing agents developed over the past few years have played an important role in shortening hospitalization. One Veterans Administration report cited shows that about half of its patients under psychiatric care are receiving tranquilizing drugs "with a remarkable shortening of acute phases of illness" in many of them.

However, despite new chemical agents and improved treatment programs, mental illness still looms as one of our greatest U. S. health problems. About one out of every 10 Americans have some form of mental or emotional disorder requiring treatment. Emotional complications are an important factor in about 50 per cent of complaints voiced to the general practitioner and 30 per cent of physical ailments treated in general hospitals.

Schizophrenia ranks as the leading cause of hospitalization for mental illness, accounting for 22.6 per cent of all first admissions, according to *Patterns.* Next are senile psychoses, which total 20.8 per cent. Severe alcoholism is the third leading single cause, with 13.7 per cent.

People in the upper social and economic bracket have the lowest rate of psychoses and the highest rate for neuroses, with men outnumbering women in that bracket in both types of mental illness.

Resection of the Temporal Lobe in Patients with Convulsive Disorders

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THIS REPORT concerns the surgical removal of a major portion of the temporal lobe in an attempt to control convulsive seizures arising in this area. Material for this study consists of twenty-five of a series of fifty patients who have been operated upon for temporal lobe epilepsy. There were twenty-one males and four females. The study was started in 1949 and only those operated upon prior to 1954 are included. To include those done subsequently would permit too short a followup period. In this series of patients the type of excision purposely has been made quite uniformly so that eventually a valid comparison could be made with results obtained with less extensive types of resection and with results of others who have excised areas comparable to that presented here.[1,2,3,4]

The temporal lobe of the brain functions as the cortical center for olfactory, auditory, taste, and vestibular impulses. It is also concerned with the storage of memory patterns, interpretation, and experience derived from auditory and visual learning and with some of the elements of emotion and their expression. This complexity of functions, and likewise of the symptoms produced by lesions of the temporal lobe, is a result of the evolutionary molding of the brain so that incorporated and compressed into it are parts of the visual, auditory, equilibratory, olfactory, taste, motor, limbic, and autonomic systems. The chief clinical feature that focuses attention to the temporal lobe is that form of seizure pattern known as a psychomotor seizure. This and the older terms of psychic equivalent and epileptic equivalence have recently been replaced by the more inclusive but even less descriptive term "temporal lobe epilepsy." The latter has originated not only as a substitute for the term psychomotor but also as an all-inclusive term for other seizure patterns arising from the temporal lobe, such as, masticatory and uncinate attacks. Thus the term "temporal lobe epilepsy" embraces the psychomotor seizure itself, in addition to others originating within the temporal lobe.

The patients in this series were selected for surgery on the basis of having: (1) clinical evidence of temporal lobe seizures without control by the usual anticonvulsant medications and (2) assaultiveness as a conspicuous part of the seizures. Although this was not an indication for surgery, it is remarkable that approximately 70 per cent of the patients exhibited abnormal personality structures and had been followed preoperatively in the University Hospitals Psychiatric Clinic. After the patients were selected for surgery they were admitted to the hospital where the usual physical and neurological examinations were carried out. Further studies included skull roentgenography, angiography, pneumoencephalography, electroencephalography, and a battery of psychological tests. Invariably the electroencephalographic studies included a series of recordings made on successive days. This was necessary to be certain of lateralization of the focus. Even so in three of the patients lateralization of the abnormal area could not be made from scalp electrography. In these instances fine copper wire electrodes were inserted through trephine holes in the skull into the depths of the temporal lobe. Electrodes were left in place over a period of four to five days during which time recordings were taken. A definite lateralizing focus was evident in each instance.

The operative procedure was usually carried out under general anesthesia although a few were done under local anesthesia. The craniotomy flap was placed to expose the major part of the involved hemisphere. Cortical electrographic studies were carried out with recordings being made on an eight channel recording electroencephalograph. A preliminary survey of cortical potentials was made covering all exposed lobes and, following

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this, individual lobes were separately scanned and a rather elaborate search was made for specific foci. Contrary to the importance placed by some authors on the tip of the temporal lobe, the majority of electrical abnormalities in this series were recorded from the lateral surface of the temporal lobe and particularly from the midtemporal gyrus. Depth electrography was performed in ten instances and was done only if excision of the lobe were anticipated. Abnormalities in the hippocampus or uncus usually were associated with abnormalities in the middle temporal gyrus. It would appear from this that a focus of electrical abnormality within the substance of the temporal lobe may spread with ease to the surface electrodes. After the confines of electroencephalographic focus were felt to be delineated, resection *en masse* of that portion of the temporal lobe was performed. Hemostasis was obtained with silver clips, little or no coagulation was done on the part of the brain left *in situ*. Resection was carried perpendicular to the surface of the brain, through the lateral ventricle, to the medial aspect of the temporal lobe so that the insula was visualized in its entirety. The free edge of the tentorium was visualized. In most instances the posterior margin resection was 7 to 9 centimeters from the tip of the temporal lobe. Electrodes were then reapplied and electrographic recordings made along the margin of the excised area. When necessary, further excision was made until no electrical abnormalities were evident. In one instance it was necessary to extend the posterior edge of the resection 14 centimeters from the tip of the lobe. Postoperatively all patients were placed on dilantin grains 1½ and mebarol grains 1½ t.i.d. The convalescent period was usually uneventful. The only complication was occasional accumulation of cerebrospinal fluid in the area of the resected temporal lobe. This was evidenced clinically by postoperative hemiparesis and increased intracranial pressure which was relieved by aspiration of cerebrospinal fluid.

Complications

One patient died two weeks postoperatively from a transfusion reaction. Two patients were aphasic for three to four weeks postoperatively but this cleared up rapidly, and they are now normal in all phases of speech.

In four patients a homonymous hemianopsia and in four others a quadrantic visual field defect was present postoperatively. Two patients had transient postoperative hemiparesis and two developed a moderately severe and persistent hemiparesis.

Results

Results of therapy, whether medical or surgical, are difficult to evaluate since so many factors enter into the course of any disease. This is especially true of convulsive disorders which are subject to many nonmedicinal influences. The chief difficulty lies, therefore, in determining the point of reference, since results are relative. For example, the word "control" is difficult to assess when applied to convulsive disorders since there is little agreement as to what constitutes good control. Ideally, control should mean complete freedom from seizures, but in practice this is a difficult goal to reach and in the literature there is no unanimity of definition for it may range from a moderate to mild reduction of seizures. Table I shows the results of surgery on the frequency of seizures. Sixteen of the twenty-five patients had, in addition to the temporal lobe seizures, attacks of other types (grand mal, petit mal) that are not considered temporal lobe in origin. Of the twenty-four patients who survived the operation, twelve (50 per cent) have been free of seizures of all types. In no instance did the operative procedure aggravate the frequency or severity of the pre-existing seizure pattern. Furthermore, a total of nineteen (79 per cent) have been relieved of their psychomotor seizures which were the chief indication for the temporal lobectomy.

TABLE I. RESULTS OF TEMPORAL LOBECTOMY IN TWENTY-FIVE PATIENTS WITH TEMPORAL LOBE EPILEPSY

Results	Number	Per Cent
Mortality	1	4
Number patients free of seizures of any type	12	50
Number patients free of temporal lobe seizures	19	79
Number patients with persisting temporal lobe seizures	5	21

Careful postoperative personality studies were done in nine of the twenty-five patients. Two of these were more disorganized following surgery, three unchanged, and four improved. More detailed data are presented in another report.[5]

Pathological Data

The lesions encountered in this series of twenty-five patients are shown in Table II. Sixty per cent showed some pathological alteration. The high percentage of tumors in this group is noteworthy. This is further worthy of attention in view of the fact that in no instance was a tumor clinically suspected or demonstrated either on plain skull roentgenography, angiography, or pneumoencephalography.

TABLE II. MICROSCOPIC FINDINGS IN TWENTY-FIVE PATIENTS WITH TEMPORAL LOBE SEIZURES

Appearance		Number	Per Cent
Degenerative changes		9	36
Neoplasms		6	24
Astrocytoma	3		
Glioblastoma multiforme	1		
Oligodendroglioma	1		
Hemangioblastoma	1		
Normal		10	40

Degenerative lesions, microscopically determined, were present in nine patients and included increase in glial cells, cyst formation, glial nodules, and gliosis, demyelinization and neuronal degeneration. These changes are difficult to evaluate and their significance is speculative, but the high percentage of apparent pathology might well explain the ineffectiveness of anticonvulsant drugs in this form of epilepsy.

Discussion

The chief clinical manifestations of temporal lobe dysfunction are epileptic and psychiatric. The former, characterized by aura, automatism, and amnesia, is the most specific indicator of temporal lobe disease. The latter is not specific in itself but when accompanied by seizures may indicate a temporal lobe lesion. It is not possible to prove at the moment that one lesion is the cause of both symptom complexes although there is considerable evidence that favors such a hypothesis. The relationship between the seizure and the psychiatric symptoms cannot be denied since the seizure itself may be manifest by rage, fear, assault, depression, disturbed visual and aural perceptions, while the interseizure aspect is characterized by change in personality structure varying from the neurotic to the overt psychotic. The temporal lobe seems to be the brain area wherein some etiology, be it tumor, injury or infection, sets in motion forces which lead to both seizures and abnormal behavior. Similar pathological lesions occurring in other brain areas are not as consistently accompanied by either seizures of this type or personality disruptions. It is felt that much of the personality aberration heretofore attributed to the frontal lobe originates in the temporal lobe. Alterations in function of the frontal lobe predispose to changes in personality of a superficial nature—inappropriate emotional tones, rather than to deeply seated severe personality disorders observed with temporal lobe dysfunction. Whether or not such personality alterations result from a lesion within the temporal lobe, or are due to a lesion elsewhere which projects through the temporal lobe and hence is altered by surgical interruption of the tracts, is not clear.

The electrographic signs of temporal lobe epilepsy are not as helpful as first anticipated. Indeed, electrography from scalp electrodes may inject confusion rather than clarification. The fact that the brain is a volume conductor of electrolytes helps to explain the discrepancies and idiosyncrasies of scalp recording. This is of sufficient importance from a practical standpoint that the clinician should never rely on a single electrographic tracing in such cases, particularly when the tracing is normal, borderline, or bilaterally abnormal. Multiple recordings are considered necessary and the value is enhanced by the use of activating procedures.

The results of extensive temporal lobe excision in the manner described in this report are gratifying from the success standpoint in the control of the psychomotor seizure itself. There are deterring factors of a potential operative mortality as well as some morbidity of a neurological and psychiatric nature. It is felt these might be decreased greatly by initial rejection of patients with known and overt deterioration. It is also possible that removal of such a mass of tissue is not the final answer to the problem. Further investigation, particularly with implanted electrodes into the deeply situated limbic system structures, may provide further knowledge and offer a better therapeutic approach to this problem.

Summary

Twenty-five individuals with temporal lobe seizures not controlled medicinally have been

(Continued on Page 380)

The Management of Tetanus

With Special Reference to the Use of Curare

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BECAUSE of the increasing use of tetanus toxoid to produce active immunity and the shift from a rural to a preponderantly urban population, the incidence of tetanus has been greatly decreased during the past thirty years. An annual average of only 500 cases is reported in the United States for the five-year period ending in 1952.[1] A review of the past ten years' records of three Minneapolis institutions, Minneapolis General Hospital, Abbott Hospital, and Northwestern Hospital, reveals a total of only five cases. During approximately the same period, twenty-six cases were reported at the Mayo Clinic[2] and thirty-two at the University of Minnesota Hospital.[3] This infrequency of occurence may easily cause physicians to overlook the possibility of tetanus in studying a patient, and such oversight may prove serious, since early diagnosis is an important element in successful treatment of the disease. With the mortality rate still reported at about 30 per cent,[1] it is essential that as clinicians we bear in mind the fact that tetanus has not yet been wiped out. Once the possibility of the disease has been considered, the differential diagnosis causes little difficulty.

A review of the literature on the therapy of tetanus reveals an area of general agreement. The consensus of opinion is that antitoxin (the usual dose in adults is 200,000 units intravenously) should be used to neutralize the toxin unless this is inadvisable because a preliminary test has demonstrated sensitivity to horse serum. If sensitivity exists, desensitization will have to be carried out before the total dose is given. The use of intrathecal antitoxin is not recommended. The surgical care of the wound, including debridement, should not be undertaken until after the antitoxin is given; and Spaeth states that the care of the wound should be based on the same principles of surgery as would be used if tetanus were not present.[4] If the patient's condition warrants the procedure, tracheotomy should be performed to provide an unobstructed airway. This also makes possible aspiration of secretions from deep in the bronchial tree and thus prevents atelectasis and pneumonia. Because it inhibits the growth of the clostridium tetanus and helps to prevent secondary pulmonary infection, penicillin is used in doses of 800,000 to 2,000,000 units a day. It is also well agreed that fluid, electrolyte, and nutritional balance should be maintained. The importance of good nursing care and, at times, constant supervision by the physician is emphasized. Here the agreement ends, with the reader left in a state of confusion concerning control of the tetanic spasms which are the cause of the patient's exhaustion and which may lead to asphyxiation.

To evaluate the effectiveness of any therapy for tetanus, Pratt[5] points out, it is necessary to evaluate the seriousness of the illness in each case. He considers four factors: the length of the incubation period, the rapidity of appearance of full-blown symptoms after initial onset, the appearance of the patient on admission, and the frequency and intensity of spasms under sedation. These facts must be kept in mind when we attempt to evaluate the reports in the literature and when we choose the type of therapy for any isolated case. No one in private practice today will see enough cases so that he will be able to draw conclusions from his own experience.

Death in tetanus is generally believed to result from three causes—exhaustion, circulatory failure resulting from demands made upon the heart, and finally asphyxia caused by spasm of the glottis, intercostal muscles, and diaphragm. A. B. Baker[6] points out that tetanus toxin has an affinity for the central nervous system, and that if the illness is prolonged there is actual cellular damage in the cerebral hemispheres and at times marked medullary involvement which may account for some cases of respiratory and cardiac failures. These findings are in agreement with those of Abel, Firor,[7] and others who studied the patho-

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genesis of tetanus over a period of years at the Johns Hopkins Hospitals, and repeatedly point out that tetanus toxin is carried by the lymphatics and blood vessels to the central nervous system, where it is fixed.

There are two fairly well-divided schools of thought regarding the choice of treatment to control the muscular spasms of tetanus. While they agree that the purpose of treatment is to keep the patient alive and his vital functions intact until the effect of the toxin wears off, they are definitely opposed as to the method. It has been the accepted practice for many years to control the spasms in tetanus with large doses of barbiturates, paraldehyde, chloral hydrate, or tribomoethanol (Avertin); and the traditional use of these drugs still persists. While these medications undoubtedly spare the patient considerable anxiety, they act as depressants on the respiratory center and may accentuate the injury of the already-damaged central nervous system. The proponents of the use of these sedatives assert that the myoneural blocking agents are too dangerous and uncertain to warrant their use.[8]

The physicians opposed to the use of the customary depressants, following a suggestion of Claude Bernard, have employed curare and curariform drugs to minimize the severity of the tetanic spasms and have developed a voluminous literature supporting the use of these preparations.

Curare in a crude form was used by the South American Indians to poison their arrow heads. The Indians classified curare according to the container in which it was stored: tube curare, pot curare, and calabash curare, each of which contains different alkaloids.[9] Tube curare has been investigated rather throughly. A physiologically-active alkaloid called d-tubocurarine chloride was isolated in crystalline form from tube curare in 1935, and this substance is the curare preparation now in general use. Very little information is available concerning pot curare and calabash curare.

Curare has been used as a general term that includes all drugs acting in the vicinity of the myoneural junction. It is sounder to refer to these agents as muscle relaxants or neuromuscular blocking agents, since some of the newer ones are not similar to the original curare. The neuromuscular blocking drugs can be classified as depolarizing neuromuscular blocking and non-depolarizing neuromuscular blocking agents, depending upon their effect at the myoneural end plate.[10] Little more is known now about the action of curare at the myoneural end plate than Claude Bernard knew in 1850, but the preparations available today make control of the results much more precise.

Aqueous preparations of curare have been administered intravenously for almost a century, and for ten years preparations of curare in wax and oil have been available for use. During the past few years, a large number of cases successfully treated with both preparations have been reported. In addition, there are several reports of cases treated with other neuromuscular blocking agents, particularly flaxidil and succinylcholine.[11,12,13,14] These drugs have been employed in extremely serious cases necessitating the use of positive pressure respirators and tracheotomy.

The principal pharmacological effect of curare is a blocking of the myoneural junction with a resulting flaccid paralysis of the skeletal muscles. The effect of subparalytic doses of curare has been pointed out by many workers as decreasing or abolishing abnormal neuromuscular responses; thus, curare in smaller doses acts as a differential screen at the myoneural junction, permitting normal impulses to pass but blocking abnormal impulses. It should be noted that curarization to the point of paralysis is neither necessary nor desirable in achieving this differential blocking action. The use of the repository type d-tubocurarine gives this subparalytic effect with the resulting relaxation of the tetanic spasms of tetanus. The use of myoneural blocking agents to a point of total paralysis, as described by Van Bergen,[11] with breathing maintained with the aid of a respirator, such as the new mobile unit developed at the University of Minnesota Hospital, should be kept in mind for the more severe cases.

Toxic effects from curare have been reported by Perlstein and Weinglass[15] and are cited by many authors as the reason for not using curare. However, as was pointed out by Segar,[16] the findings of Perlstein and Weinglass are based on animal experiments in which much larger doses were employed than are customarily administered in anesthesia or in treatment of such diseases as tetanus.

Most of the reports on the use of neuromuscular blocking drugs emphasize the importance of having readily available the necessary equipment for resuscitation in event that respiration becomes embarrassed. This equipment includes prostigmine or tensilon, oxygen, face mask, breathing bag, laryngoscope, and endotracheal catheter, complete with necessary connections. If these precautions are taken the drug can be used with relative safety.

A newer drug, mephenesin, has been studied for many years by the pharmacologists, but only recently has its clinical use been reported in the literature.[17] Mephenesin produces a temporary paralysis superficially resembling that caused by curare, but apparently the result of a depressant effect on the brain stem rather than a blocking action at the myoneural junction. A sedative action is also reported and must be considered when mephenesin is used in conjunction with barbiturates or other depressant drugs. A review of several of the reports on the treatment of tetanus with mephenesin reveals that the drug is generally used along with large doses of barbiturates. In several cases the convulsions were not controlled by the mephenesin.

The use of mephenesin may have undesirable side effects. Thrombophlebitis is likely to develop at the site of repeated intravenous injections, and a hemolytic effect has been reported when a solution stronger than 2 per cent has been employed. Local anesthesia of the mucosa of the mouth and pharynx has been reported when the powdered drug is taken orally.

According to some writers on the subject, mephenesin is less dangerous and as effective as curare for muscular relaxation.[18,19,20,21] A review of the reported cases does not lead me to the same conclusion, although the drug should, of course, be a part of the physician's armamentarium.

The most recent addition to the therapy of tetanus is the use of cortisone, hydrocortisone, their newer derivatives, and adrenocorticotropic hormone. Lewis[14] and his associates report a controlled study in which, in severe tetanus, half of the patients were given A.C.T.H. or cortisone and half were not, with a marked shortening of duration and decrease in the severity of the course in the treated group. There was also a marked improvement in mortality. Their best results were obtained with oral cortisone, although the authors have no good explanation for the superiority of the oral medication. Christensen in the June issue of Minnesota Medicine[2] states that the action and value of these drugs have not been determined and suggests their use be limited to allergic patients and those in whom exhaustion becomes apparent. In these circumstances, they are employed simply as supportive measures in an attempt to maintain life while the patient is passing the crisis of this self-limited disease.

Case Reports

Case 1.—A Minnesota farmer, aged forty-six, cut his finger in the sickle of a grain harvester, July 24, 1952. He went at once to his local physician, who cleaned the wound, closed it with two sutures, and then gave him an injection of penicillin. The wound healed uneventfully. On July 31, 1952, he developed general malaise and chills. The next day, he developed muscular rigidity and intermittent generalized muscular spasms. These symptoms became progressively more severe until he was brought to the Abbott Hospital, August 4, 1952. Examination on admission revealed the patient to be awake and mentally alert. There was a generalized muscular rigidity, and he was unable to open his mouth more than 1 cm. There was a marked opisthotonus and a board-like rigidity of the abdomen. Blood pressure was 128 systolic and 52 diastolic. The pulse was 78 and of good quality. The respirations were 20 a minute and appeared to be forced. Every ten or fifteen minutes, and, following the slightest stimulus, the patient had painful tetanic convulsions that made him weep. These lasted one to two seconds, and after the convulsions, he had difficulty breathing. The wound on the left index finger was well-healed with no evidence of infection. The leucocyte count was 13,350/cu. mm. with 83 per cent neutrophils. Urinalysis showed 1 plus albumin and no other abnormal findings.

Following the negative sensitivity to horse serum test, the patient was given 120,000 units of tetanus antitoxin intravenously. He was also given 1.5 cc. of tetanus toxoid subcutaneously and sodium phenobarbital by hypodermic in doses of 300 mg. for sedation. Penicillin, 800,000 units daily intravenously, was also started.

Ten hours after admission, in spite of two doses of 300 mgs. (5 grains) of phenobarbital, his condition was unchanged. At this time he was given the first injection of 25 mgs. of tubocurarine in oil and beeswax. Within twenty minutes he was visibly more relaxed, and thirty minutes after this injection he fell asleep for the first time since admission. He slept for an hour. Two hours after the curare was given, he asked for a drink of water. He choked on the second swallow and had a severe convulsion. During his second hospital day he was given 2400 mgs. of phenobarbital (40 grains) in a 24-hour period. On August 6, 1952 at 8 a.m. he was given 25 mgs. of repository tubocurarine, and within ten minutes there was improvement in the muscular rigidity. A nasal tube was passed without producing convulsions, and only 1380 mgs. of sodium phenobarbital was needed in this 24-hour period. On August 6, 1952, A.C.T.H. was also started, 10 units being given intramuscularly every six hours. The repository tubocurarine was given every forty-eight hours, and the sodium phenobarbital was gradually decreased until on August 11, 1952, he had only 180 mgs. in twenty-four hours. On August 14, 1952, the gastric feeding tube was removed. The tubocurarine and sodium phenobarbital were discontinued, and only chloral hydrate, 10 to 15 grains, was administered as needed for sleep. His subsequent course was uneventful, and he was discharged from the hospital August 24, 1952, three weeks after admission.

Case 2.—The second case is that of a Minnesota farmer, aged thirty-five, who apparently was well and gave no history of injury prior to the onset of illness, April 30, 1953. He developed generalized muscular stiffness, and when he retired that evening fell out of bed onto the floor and was unable to get up. For over forty-eight hours he lay on the floor, where he was found May 2, 1953, by a brother who lived on the next farm. He was taken to the local hospital and then brought to Minneapolis, May 3, 1953.

Examination on admission to the Abbott Hospital revealed a well-developed, well-nourished white man who exhibited such marked generalized muscular rigidity that he was unable to move his arms and legs. His neck was stiff. Although awake and conscious, he could not talk because trismus prevented his opening his mouth more than 1 cm. Any stimuli produced generalized convulsions. He had extremely poor oral hygiene. His pulse was 112; respirations, 20; temperature, 102.6°; and blood pressure, 140/92.

Following sensitivity to horse serum tests, he was given 80,000 units of tetanus antitoxin intravenously and 40,000 units intramuscularly. Penicillin, 400,000 units, was started twice daily intramuscularly. Shortly after admission he was given 25 mgs. of repository d-tubocurarine intramuscularly. Four hours later he was able to move his extremities and to swallow water. There was marked relaxation of the generalized rigidity and the tetanic convulsions ceased. He was given sodium phenobarbital, 300 mgs. intramuscularly, for sedation at 8 p.m., and this was repeated five hours later at 1 a.m. During his first twenty-four hours in the hospital, one of his physicians was in constant attendance. Thereafter, the patient was given only 300 mgs. of sodium phenobarbital at bedtime each day for his first ten days in the hospital, and his muscular spasms were controlled well with repository d-tubocurarine, 25 mgs. every twenty-four to thirty-six hours. The last dose was administered at 7 a.m., May 14, 1953, eleven days after admission. The total intake of d-tubocurarine was 175 mgs., given in seven doses. The patient walked out of the hospital without assistance, June 1, 1953.

Case 3.—A white man, aged seventy-seven, developed an infection in the palm of the left hand secondary to a puncturing wound by a splinter of wood from an old ladder about May 8, 1953. A few days later he developed generalized stiffness, and on May 15, and May 16 his sister removed parts of the sliver. About May 21 he was having some stiffness in his jaw. Attributing this to poor teeth, he saw a dentist, who removed two teeth. At that time it was hard for him to open his mouth. The generalized stiffness gradually progressed, and he developed difficulty walking. About June 1 he had trouble breathing and for the first time he was seen by his physician, who advised hospitalization. The patient was sent to a private hospital June 2, 1953 and then transferred to Minneapolis General Hospital at 10 a.m., June 3, 1953. A more detailed history was not possible because both the patient and his sisters were very uncertain about the sequence of events.

Examination on admission to Minneapolis General Hospital revealed a well-developed, well-nourished elderly white man who exhibited generalized muscular rigidity and a mild opisthotonus. He was unable to open his mouth and his facial muscles were described as being in severe spasm. There was poor oral hygiene and some bleeding from the mouth, which could not be opened to determine the source of the blood. There was marked neck rigidity. The temperature was 101° F; the pulse, 112; the respirations, 24; and the blood pressure was 160 systolic and 100 diastolic. He was having severe tetanic convulsions following even mild stimuli. Emergency laboratory studies were within normal limits.

After a test for sensitivity to horse serum was found to be negative, 120,000 units of tetanus antitoxin were given intravenously; and penicillin was started in doses of 300,000 units every eight hours. He was given 20 cc. of paralydehyde by rectum. Two and one-half hours after admission, under intravenous surital and curare, the patient was put to sleep, and a tracheotomy was performed. The indurated area in the left palm was excised and the tissue cultured for clostridium tetanus, but none was found. During the next twenty-four hours he had a rather stormy course and his tetanus became more severe. Consequently, June 4, 1953, he was given an additional 250,000 units of tetanus antitoxin intravenously.

At 5 p.m., June 4, 1953, the first 25 mgs. dose of repository d-tubocurarine was administered. It produced marked muscular relaxation. The tubocurarine was repeated at 9:15 a.m., June 5 and whenever needed to maintain muscular relaxation until June 10, 1953, when it was no longer necessary. He was given a total of 300 mgs. over a six-day period with no serious side effects. His subsequent course was uneventful, and he was discharged from the hospital June 22, 1953. When he was seen June 25, 1953, he was well.

Summary

Although through the increasing use of tetanus toxoid the incidence of tetanus is declining it is desirable to continue to bring this preventable disease to the physician's attention. It should be pointed out that while the value of tetanus immunization is generally accepted, there are still many people who are without this protection. Somehow this situation should be publicized; and an educational plan should be developed, directed to lay people and physicians, especially those serving a rural population, to encourage the use of tetanus toxoid immunization and booster injections, much as the pediatricians are administering them to children in their routine practice.

The various drugs used to control an active case of tetanus are reviewed, with emphasis on the use of the myoneural blocking agents to manage the tetanic convulsions.

Three cases of tetanus are reported that were treated with repository d-tubocurarine with favorable results.

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RESECTION OF THE TEMPORAL LOBE

(Continued from Page 375)

treated by a uniform type surgical excision of the major portion of the temporal lobe.

Seventy-nine per cent of the patients have had cessation of the psychomotor seizures during a followup period varying from two to seven years.

Fifty per cent of the patients have been free of seizures of any type during the followup period.

Fifty per cent of the patients have continued to have either major or minor seizures (non temporal lobe).

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The Use of the Chromatin Smear in Gynecology

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SINCE the recent discovery that genetic sex can be determined by the simple examination of vaginal and buccal smears it has become necessary to change some of our previous concepts regarding problems of intersex. The chromatin smear has greatly simplified diagnoses in some of these problems. It has reduced the necessity for exploratory laparotomy in some cases, and has helped to prognosticate in the field of infertility.

For those of you who are not using this aid, I would like to outline the types of cases in which it is extremely useful, taking two of the cases from my own practice as illustrations.

Technique

We have tried a variety of staining solutions and prefer cresyl-echt-violet stain as originally described by Moore and Barr.[1] Buccal smears are more easily obtained than vaginal smears and are usually less contaminated with bacteria. It is also possible for one to use Papanicolaou stains for the study of the chormatin pattern. This is extremely valuable in cases where one wishes to review old slides to study the chromatin pattern on patients on whom the Papanicolaou smears have already been taken. As a routine, however, the Papanicolaou technique has not been as satisfactory as the cresyl-echt-violet.

In the genetic female the nuclei of the mucosal cells contain a mass of deeply staining chormatin about one micron in size (Figs. 1-4). This is usually located at the periphery just under the nuclear membrane. In the preparation of the smear the highest technical quality is required. Several smears should be made in order to have one of proper thickness. A common fault is making too thin a smear, inasmuch as one hundred cells have to be counted and a large number are necessary. If 40 to 50 per cent of the nuclei of good technical quality contain the chromatin mass this may be interpreted as a chromosomal female or preferably chromatin positive. Less than 6 per cent is interpreted as a chromosomal male or chromatin negative. Inasmuch as the appropriate sex of a patient with a congenital error may not conform with the chromosomal sex, it is better to use the term chromatin positive and chromatin negative, rather than male or female.

Classification of Intersex Problems

A pseudo-hermaphrodite is an individual with male or female gonads and some degree of ambisexual differentiation of the accessory sexual structures. They are either male or female pseudo-hermaphrodites depending upon the morphology of the gonad. A true hermaphrodite, a rarity of which only approximately sixty cases have been reported in literature, will possess both ovarian and testicular tissue. The term "hermaphrodism" has recently been broadened to include cases of gonadal dysgenesis where the gonads are faulty or absent, and in which chromosomal smears do not agree with the somatic development.

Female Pseudo-hermaphrodism

Congenital adrenal hyperplasia is the commonest type of female pseudo-hermaphrodism. It is a familial disorder of the adrenal in which there is an excess of adrenal androgens resulting in an elevated 17-ketosteroid excretion and increasing pregnantriol. The internal genitalia are female. The chromatin pattern is female, but the external genitalia are usually abnormal with enlarged clitoris and some fusion of the labial-scrotal folds. One-third of these cases have an accompanying adrenal cortical insufficiency and in infancy may exhibit severe Addisonian-like electrolyte disturbances. Virilization occurs along with accelerated skeletal growth. The diagnosis in these patients should be made as early as possible so that treatment with cortisone will prevent virilization and rapid growth. Clitorectomy and plastic perineal

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Presented before the Minnesota Obstetrical and Gynecological Society, November 30, 1957.

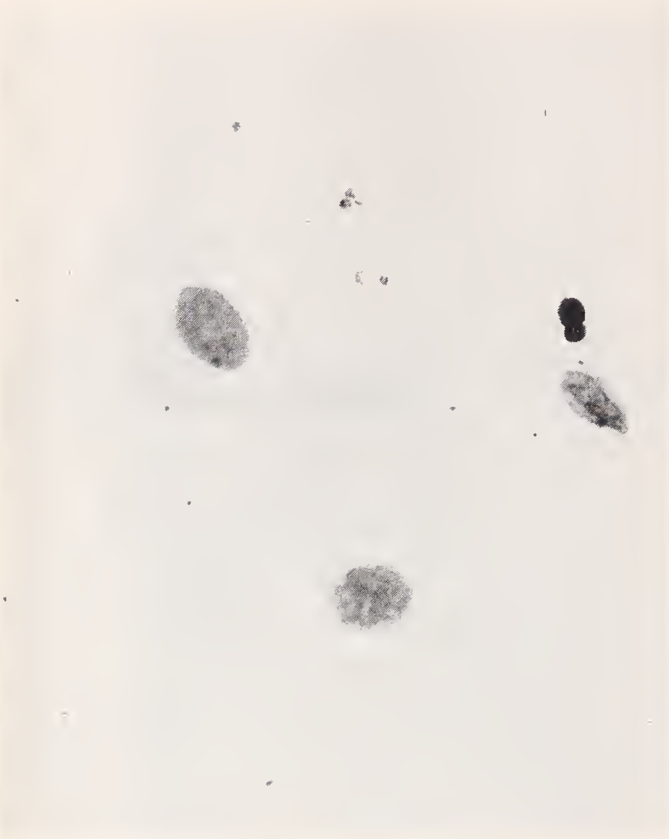

Fig. 1. (x 1040) Buccal smears showing typical female chromatin pattern (cresyl-echt-violet).

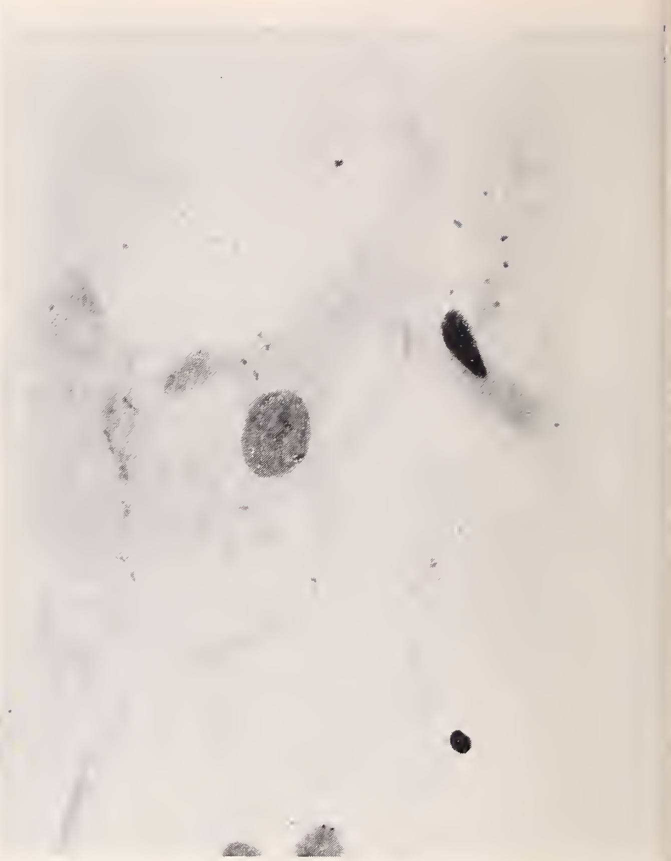

Fig. 2. (x 1040) Same as Figure 1. Chromatin mass underlying nuclear membrane (cresyl-echt-violet).

repairs are usually indicated. Differential diagnosis includes true hermaphrodism and male hermaphrodism with bifid scrotum and undescended testicle. The use of the chromatin smear readily eliminates the latter. Elevated 17-ketosteroid excretion differentiates it from the second type of female pseudo-hermaphrodism, namely, female pseudo-hermaphrodism without adrenal hyperplasia.

Case 1 (J. M.)—An eleven-year-old girl was seen in 1956 primarily because of the enlarged clitoris as well as hypertrichosis of the thighs and chin. History obtained was that an enlarged clitoris and labial-scrotal fusion were noted at birth. She had been examined at the University Hospital at the age of five years because of the enlarged clitoris and pubic hair; 17-ketosteroid excretion at that time was 7 mgm/24 hrs. and F.S.H. 4 M.U./24 hrs. Diagnosis at that time was: (1) adrenal hyperplasia, (2) adrenal tumor, or (3) gonadal tumor. Chromatin smears, of course, were not known at that time (1950). Laparotomy was advised, but was refused by the parents. Six years later her 17-ketosteroid excretions were 24.5 mgm/24 hrs. and 23.9 mgm/24 hrs. Chromatin smears were positive. A clitorectomy was performed (clitoris was 4 x 1½ cm.) and the patient was placed on 50 mg. of cortisone a day. The 17-ketosteroid excretion level dropped to 10.2 mgm/24 hrs. Hypertrichosis and acne have disappeared, and the patient is now menstruating.

Female hermaphrodism without adrenal hyperplasia is the rarest form of intersex and may be produced by the administration of testosterone or progresterone to the mother, or by a maternal arrhenoblastoma. The genitalia are similar to the type associated with the adreno-genital syndrome just described. There are sometimes associated urinary tract anomalies which may produce a recurrent hematuria due to menstruation. Contrary to the adrenal hyperplasia group, these patients menstruate and develop female secondary characteristics. The 17-ketosteroid level, of course, is normal. Howard Jones[2] recently reviewed this condition in one of the recent journals and found a total of twenty cases. One was due to an arrhenoblastoma, three were exposed to testosterone given to the mother prenatally, and three were exposed to progesterone. The last group is extremely interesting to the obstetricians due to the frequency with which progestational drugs are used in early pregnancy. Howard Jones believes

Fig. 3. (x 1040) Proper interpretation impossible because of overlying bacteria (cresyl-echt-violet).

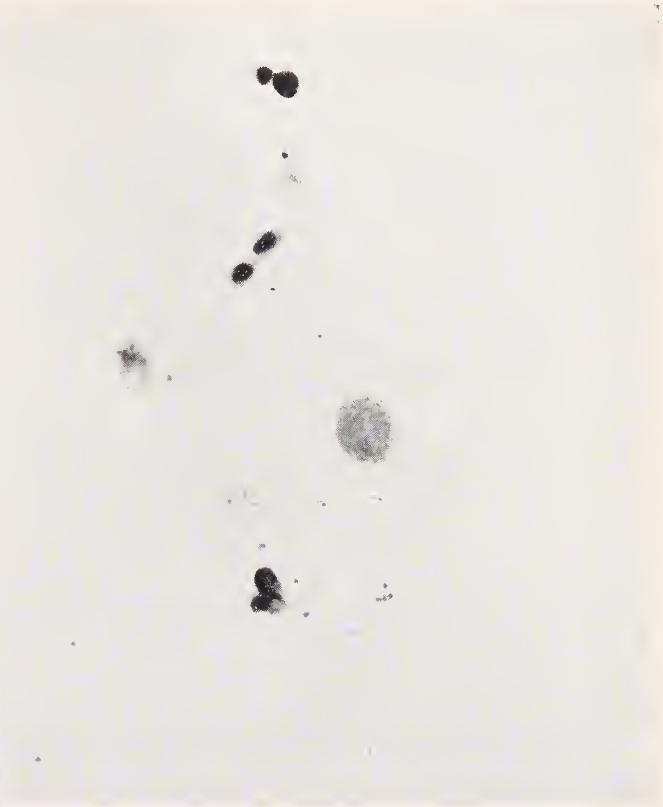

Fig. 4. (x 1040) Buccal smear from Case 2 (gonadal dysgenesis). Negative chromatin pattern.

that when embryonic genitalia are in the formative stage, progesterone should be used only when there is a demonstrable deficiency and then in physiological amounts. Diagnosis of this type of female pseudo-hermaphrodism depends upon the exclusion of progressive virilization as evidenced by a normal 17-ketosteroid level. The female chromatin pattern is noted. True hermaphrodism must be excluded by laparotomy.

Male Hermaphrodism

Male hermaphrodism is divided into two groups—male and female, depending upon the anatomy of the external genitalia. In both, the gonads are testes and the chromatin pattern is male in character. The female variety, or so-called testicular feminization, is a familial condition which is often unsuspected and is relatively common. Outwardly the individual is completely feminine with well-developed breasts and prominent areoli. There is a rudimentary vagina with usually absent internal genitalia. Male gonads are frequently found in an inguinal or labial hernia. There is an increased F.S.H. (follicle stimulating hormone), normal 17-ketosteroid excretion, and, of course, negative chromatin smears. While the fetal testes may produce female secondary characteristics, they should be removed because of the danger of malignancy. Estrogen therapy is indicated.

In congenital absence of the vagina a positive chromatin smear excludes the testicular feminization syndrome. In the latter, the testes are secreting estrogen and the vagina may be shortened or of normal length, but ends blindly with an absence of the uterus. A chromatin smear will serve to differentiate the two conditions, and it is extremely important that this be done, inasmuch as gonadectomy is indicated in testicular feminization because of the likelihood of malignant degeneration.

In the second group of male hermaphrodites, the external genitalia are ambiguous or male in type, with varying degrees of ambisexual development. There is commonly perineal hypospadius and cryptorchidism.

True Hermaphrodism

True hermaphrodism is a rare condition with approximately sixty cases reported to date. Such patients have gonads of both sexes either as separate ovary or testis, or combined as an ovi-testis.

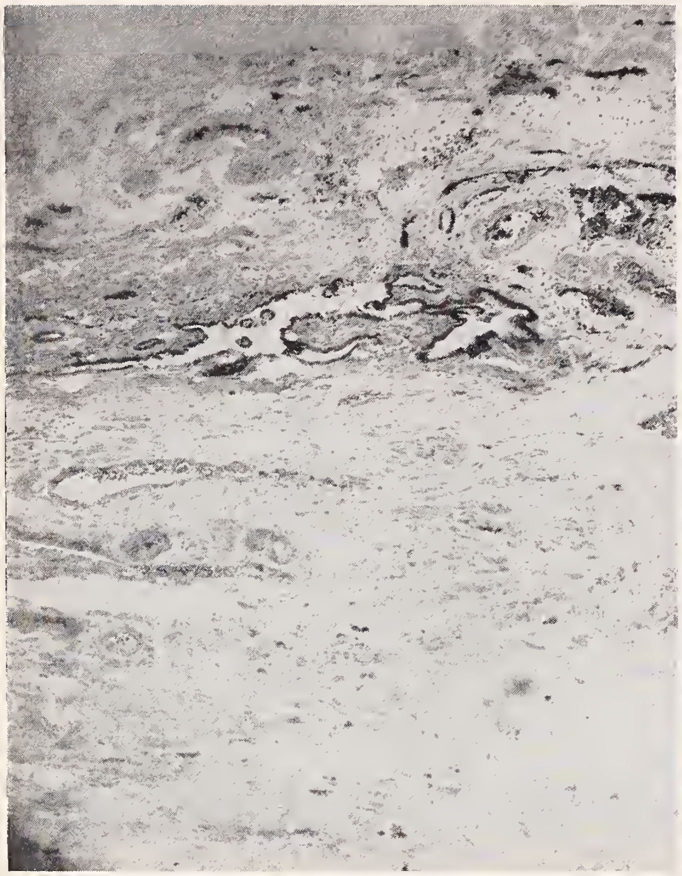

Fig. 5. (x50) Gonadal tissue from Case 2 (gonadal dysgenesis). Note total absence of primordial follicules, predominance of stromal tissue, and well-defined rete ovarii.

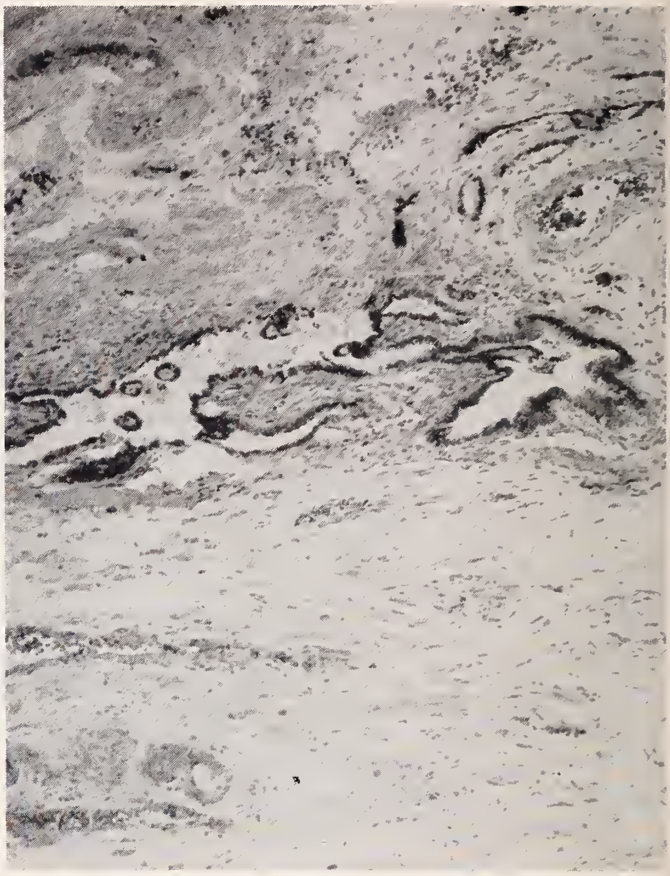

Fig. 6. (x 100) High-power view of rete ovarii from Figure 5.

The sex chromatin pattern is usually female. True hermaphroidism is accurately diagnosed by laparotomy alone.

Gonadal Dysgenesis

Turner[3] first described his syndrome in 1938. This clinical picture consists of primary amenorrhea, sexual infantilism, dwarfism, slight to moderate delay in bone age, osteoporosis, high F.S.H. excretion, congenital anomalies, such as webbing of the neck, cubitus valgus, and coarctation of the aorta.

Recently, Polani et al,[4] surprised by the rather high incidence of coarctation of the aorta in these cases, discovered that three of their so-called Turner's syndrome patients were genetically male. This was corroborated by Wilkus who discovered that twenty out of twenty-two such patients were male. This paradox has been explained by the work of Jost and Raynaud which showed that early castrated fetuses of rodents developed as female regardless of the genetic sex. The term gonadal dysgenesis is therefore applied to this condition, and our present concept is that the germinal epithelium of the fetal testis is damaged before the ninth week of embryonic life. Because of the absence of fetal testicular tissue, the embryo becomes feminized and male characteristics are suppressed. The reason for higher incidence of male over female as given by Grumbach[5] is "that the fetal testis embryologically differentiates before the ovary. The male gonad, particularly its germinal elements, may be more susceptible to damage from some unknown cause in early intrauterine life than the fetal ovary."

The diagnosis of this condition is established by finding negative chromatin smears in individuals with primary amenorrhea and usually short stature. There may be an elevated follicle-stimulating hormone excretion. However, in some patients this does not appear until puberty or later. Laparotomy will reveal a tiny uterus, redundant tubes, and so-called "streaking" of gonadal tissue below and running parallel to the tubes. Therapy consists of administration of cyclic hormonal therapy to help develop secondary sexual characteristics.

Case 2 (E. S.)—In 1947, a twenty-five-year-old single woman was seen because of primary amenorrhea and pruritus vulvae. Physical examination revealed her height to be 57¼ inches, span 60½ inches, weight 93 pounds, and blood pressure 114/80. There were no familial endocrinopathies. There was noted a slight lisp, low hair-line, and a mild degree of webbing of the neck. There was an increased carrying angle, absence of axillary and pubic hair, and absence of breast development with broad breast shield. Urethroscopic examination of the vagina revealed a tiny cervix. Vaginal smear was castrate in character. X-rays of the sella turcica were normal and the epiphyses were closed. Administration of cyclic estrogens resulted in withdrawal bleeding and increased development of the breasts and genitalia.

In 1949, the patient married, making an entirely satisfactory marital adjustment. The cyclic hormonal therapy was continued. The pruritus vulvae was unaffected by any therapy. In 1956 leukoplakic vulvitis was rather severe. Buccal, vaginal and blood smears for chromatin were made, revealing a typical male pattern. In 1957 a simple vulvectomy was performed for vulvar leukoplakia. Laparotomy revealed findings typical of gonadal dysgenesis, namely, infantile uterus with long, thin, convoluted tubes. Gonadal streaking was seen running parallel to the fallopian tubes. Sections of gonadal tissue are seen in Figures 5 and 6.

Seminiferous Tubular Dysgenesis

Seminiferous tubular dysgenesis refers to the condition in male patients with small or atrophic testicles and azospermia due to a congenital defect of the gonads. The sex chromatin is positive or negative but is predominantly positive. Grumbach stated recently that he believed that 50 per cent of azospermic males would eventually be proven to be chromatin positive. The F.S.H. excretion is usually elevated and there may or may not be an associated Klinefelter's syndrome which is, namely, gynecomastia, eunochoidism, and small testes. There are all degrees of fibrosis and hyalinization of the seminiferous tubules, with absence of spermatogenesis.

Summary

The chromatin smear is a valuable adjunct in the diagnosis and treatment of congenital errors of sex development. For the gynecologist the buccal and vaginal smears are the most satisfactory. In addition to its use in cases of hermaphrodism, it is suggested that the smear be performed for any patient with primary amenorrhea accompanied by short stature or any failure of secondary development of sexual characteristics. Since at least 80 per cent of the cases of gonadal dysgenesis are chromatin negative, the diagnosis may be confirmed by means of the chromatin smear. If the chromatin pattern is female the diagnosis is neither confirmed nor refuted. In azospermic males the finding of chromatin positive smears confirms the diagnosis of Klinefelter's syndrome and precludes the possibility of future paternity.

In cases of adreno-genital syndrome, the chromatin positive smear and the elevated 17-ketosteroid excretion are the most important aids in establishing an early and correct diagnosis during the neonatal period. Exploratory laparotomy is no longer necessary in this condition.

In testicular feminization characterized by amenorrhea, short vagina, and negative chromatin smears, laparotomies will reveal testicular tissue and should be performed. The value of the chromatin smear in the diagnosis of this condition is obvious, and its use will probably reveal a larger incidence of this condition than has heretofore been suspected.

In regard to the therapy of these errors of sex development, it is generally agreed in the literature that the appropriate sex is not necessarily the same as the gonadal, but should be determined by the anatomy of the external genitalia. However, any attempt at alteration of sex after the age of three years is generally agreed to be extremely dangerous in regards to psycho-sexual development.

The gynecologist, of course, will see chiefly those individuals who are living as females and should continue to do so. His job, therefore, is the administration of cortisone to the congenital adrenal hyperplasias, removal of the overgrown phallus, administration of estrogens to the gonadal dysgenesis patients, and the construction of artificial vaginas. Some of the ambisexual individuals will require perineal plastic operations and hormonal therapy.

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Fetal Salvage in Rh Incompatibility Where Previous Stillbirths Have Occurred

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EARLY adequate exchange transfusion has altered profoundly the prognosis for the infant having hemolytic disease of the newborn. Even in severely affected liveborn infants, death or kernicterus following adequate treatment should be much less than 5 per cent. This excellent outlook for the infant following Rh sensitization does not apply where a previously affected baby has been a fetal hydrops or stillborn. Over 80 per cent of all Rh-positive infants born following a previous hydrops or stillborn child will be similarly affected. This has been our experience in Winnipeg and similar experience has been adequately documented by others, particularly Davies and her colleagues in Birmingham.[1] Indeed, should there have been more than one previous hydrops or stillborn the possibility of further living Rh-positive babies at term is almost zero.

Probably all fetal deaths from Rh sensitization occur after the twenty-eighth week—most of them (in our experience) after the thirty-fourth week of pregnancy. The presence of living babies *in utero* at thirty-four to thirty-five weeks in these women has not unnaturally led to the feeling that induction or cesarean section, sufficiently early to produce living premature infants on whom treatment would be possible, might lead to the salvage of some of these infants.

It is indeed unfortunate that the report of Mollison and Walker in 1952,[2] which was the first paper to give statistical proof of the life-saving effect of exchange transfusion in hemolytic disease, should have thrown early obstetrical induction into disrepute. They showed that in a random sample of infants with hemolytic disease of the newborn, the prognosis was better in the group where delivery occurred at term than in those where delivery was induced at thirty-six to thirty-seven weeks. This is not surprising when one considers that serum bilirubin levels were not followed, and that repeated exchange transfusions were not done. In the induced group induction was too late, and treatment not intensive enough to alter significantly the prognosis in the most severely affected babies. From this study and from those of Diamond and others,[3] has sprung an exaggerated opinion of the dangers of prematurity in hemolytic disease.

It is difficult to see how there can be any argument against early induction of labor where the father is homozygous Rh-positive and there have been previous fetal hydrops or stillborn infants due to Rh incompatibility. We know that, at term, the possibility of a living baby is very remote. Early induction has the virtue of holding forth some hope of a living salvageable baby and can do no harm to the infant whose prognosis is otherwise so poor. This argument has led to the practice of early delivery in these cases in several centres with encouraging results.[4,5,6] Indeed, Walker of Newcastle, who participated with Mollison in the study which originally discouraged induction has now a large series,[7] with favorable results. Surely, however, since they induced all mothers at thirty-six weeks, in many of his "failures" even earlier induction would have led to success.

We first tried really early induction in such a case in late October, 1954.

Report of Cases

This woman had a tragic but altogether too familiar history. Her first child was living and normal. Three subsequent pregnancies all terminated spontaneously at thirty-four to thirty-five weeks with stillborn macerated fetal hydrops. The prognosis for the pregnancy in 1954 could hardly have been worse. A surgical induction was performed at thirty-two weeks. Following a very worrying period of ninety-six hours of inactivity she finally went into labor. At this point, we had just about given the infant up but had not felt sufficient confidence in the outcome to advise cesarean section as we now would do after forty eight hours of failure to go into labor. However, on November 1,

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This work was done in collaboration with Dr. Bruce Chown, Director of the Blood Group Reference Laboratory, Winnipeg, Manitoba, Canada.

Presented before a meeting of the Northwest Pediatric Society, Bayport, Minnesota, September, 1957.

1954, she delivered a living female child weighing 3 pounds 11 ounces. The infant was limp and pale and had a large liver and spleen. However, she cried spontaneously and presented no problem in resuscitation. Her cord blood was Coombe's positive and showed evidence of very severe hemolytic disease indeed, with a hemoglobin of 6.7 gm. per cent and a bilirubin of 5.2 mgm. per cent, (the normal cord blood bilirubin in our laboratory being 2 mgm. per cent). A two-blood-volume exchange transfusion was performed at three hours of age and due to subsequent elevation of serum bilirubin to over 20 mgm. per cent, this had to be repeated at sixteen, twenty-six and sixty hours of age. All four exchange transfusions were done through the umbilical vein, the infant's condition remaining good throughout. She was discharged from the hospital on the thirty-eight day, free of jaundice and clinically normal. This baby was, to say the least, very encouraging. There can be no doubt that her life was saved by early induction. Now, at almost three years, she is normal in every respect.

In December of the same year we were presented with the difficult problem of a woman who had had a previous hydrops married to a heterozygous husband. During the pregnancy there had been no change in antibody titre to indicate whether the baby was Rh-positive or negative. This is usually the case in such situations. There was considerable vacillation here but finally she was induced at thirty-six weeks only to produce a fetal hydrops. One cannot help feeling that induction at thirty-four weeks might have saved the infant.

In February, 1955, our next opportunity of managing a case was presented by a mother with a bad history and married to a homozygous husband.

Here is a truly tragic story. A normal child was born in 1945, and was killed in a street accident at the age of seven; a second child suffering with severe hemolytic disease of the newborn in 1948, died at four days of age; a fetal hydrops was delivered in 1950 and again in 1952, the fetal deaths having occurred at around thirty-five weeks in both instances.

This woman had minimal antepartum hemorrhage in the fifth pregnancy in 1955 and since x-ray suggested a partial placenta praevia, a cesarean section was performed at about thirty-three and one-half weeks. A 4 pound 12 ounce baby boy was delivered. He was pale and limp, and had a huge liver and spleen. The cord blood was Coombe's positive with a hemoglobin of 6.0 gm. per cent and a bilirubin of 5.4 mgm. per cent. Two-blood-volume exchange transfusions were performed through the unbilical vein at two and one-half, thirty and fifty-four hours of age, a serum bilirubin exceeding 20 mgm. per cent being the indication, as always, for the repeated exchange transfusions. At fifteen months of age this was a perfectly normal little boy. There is no doubt that early section saved his life.

Since then we have treated successfully ten further similar cases. Time will not permit detailed consideration of each case.

All ten mothers had circulating anti D and had had at least one previous stillborn fetal hydrops—most of them had had several. The fathers were all homozygous Rh-positive. Pregnancy was terminated at thirty-six weeks in one woman, before thirty-five weeks in the nine others. Birth weights varied from 4 pounds 4 ounces to 6 pounds 8 ounces with most infants being under 5 pounds. Cord bloods were all Coombe's positive with hemoglobins from 3.7 gm. per cent to 11.8 gm. per cent, and bilirubins from 3.96 mgm. per cent to 6.8 mgm. per cent. All babies are surviving and apparently normal following early and repeated exchange transfusions.

As our ninth case was a brother of the little girl in Case 1, I should like to present him in more detail.

From the above you will remember this woman's history: a normal baby in 1947; fetal hydrops in 1948, 1950 and 1951; a surviving, now normal child in 1954 following surgical induction at thirty-two weeks and four exchange transfusions. On January 13, 1956, she was again induced at 32 weeks. Again, she did not go into labor for ninety-six hours but finally, on January 17, she delivered a 4 pound 15½ ounce baby boy. The baby was extremely pale and edematous, almost a hydrops. The cord blood hemoglobin was 6.9 gm. per cent, the bilirubin 4.5 mgm. per cent. Five minutes after delivery, respiratory distress was so severe that it seemed he was dying in heart failure. The cord was transected immediately, 50 cc. of blood was withdrawn and the exchange transfusion was begun ten minutes after delivery. The baby's clinical condition improved dramatically during the exchange and although repeated exchanges were necessary at forty-nine and seventy-three hours of age, he never gave further cause for worry. At fifteen months of age he appeared to be perfectly normal.

Comment

Thirteen cases of pregnancy in twelve women who have previously delivered at least one fetal hydrops or stillborn baby due to Rh sensitization, are presented. In one, where the husband was heterozygous, induction at thirty-six weeks was too late and a fetal hydrops resulted. In the other twelve, all husbands were homozygous Rh-positive, induction or section was performed at or before thirty-five weeks, and all the infants survived, apparently normal, following adequate exchange transfusions.

The author advocates induction or section before thirty-five weeks in all women who have had a previous hydrops whether the husband is homozygous or not. With a heterozygous father there is a 50 per cent chance of a normal Rh-negative baby at term. If the baby is Rh-positive, there is an 80 to 100 per cent chance of fetal death. There is, to my knowledge, no certain way of determining in these cases the Rh status of the baby *in utero,* although recent work suggests that colorimetric examination of amniotic fluid at thirty-two weeks may be of help.

The over-all mortality, therefore, at term is 40 to 50 per cent. Delivery at or before thirty-five weeks in our series of twelve Rh-positive infants gave 100 per cent survival. It might be argued that we were a bit lucky and, as I will show in a moment, this is probably so. However, one should be able to count on at least 80 per cent survival. Davies, Gerrard, and Waterhouse,[1] report 100 per cent survival in eleven cases similar to ours from Birmingham. The mortality from prematurity and other causes in the Rh-negative infants delivered after induction or section at thirty-five weeks should not exceed 10 per cent. This would give a total mortality of not more than 10 to 15 per cent following delivery at thirty-five weeks where the father is heterozygous. Compared to the 40 to 50 per cent mortality at term there can be no doubt about the desirability of early delivery.

From this series of twelve cases it might seem that one should expect 100 per cent survival following early delivery. I think this expectation would leave us disappointed. While it is true that we have not lost one baby of an Rh-sensitized mother (who had had a previous fetal hydrops) following delivery at thirty-five weeks or less, we have lost several babies where early delivery was performed for other reasons.

In an effort to prevent even the first fetal hydrops, for the past two years, we have been inducing labor at thirty-four to thirty-six weeks in all women showing at least a fourfold rise in albumen antibody titre in the third trimester. There have been some thirty such cases with six deaths. There was one fetal hydrops, two infants were stillborn as a result of obstetrical complications, three others (weighing approximately 4½ pounds) were very severely diseased —each surviving exchange transfusion only to die at about twenty-four hours of age with hyaline membrane syndrome. The remaining infants were all very severely affected by hemolytic disease of the newborn but survived and are normal following repeated exchange transfusions. From the hematological condition of these infants at premature delivery, it seems certain that at term a good many more than six of them would have been stillborn.

Protocols of Cases

(All mothers Rh negative. All fathers homozygous Rh positive)

Case 1.—Baby W. Previous pregnancies—1947, normal baby; 1948, 1950 and 1951, stillborn fetal hydrops. Present pregnancy—antibody 16 albumen. Surgical induction November 1, 1954 at thirty-two weeks. Birth weight 3 pounds, 1 ounce—Female. Cord blood—Hb. 6.7 gm. per cent, Bil. 5.2 mgm. per cent. Exchange transfusions at three, sixteen, twenty-six and sixty hours. At thirty months development normal.

Case 2.—Baby V. Previous pregnancies—1945, normal (killed in a street accident, age 7 years); 1948, jaundiced, died at 4 days; 1950 and 1952, stillborn hydrops. Present pregnancy—antibody 8 albumen. Cesarean Section February 12, 1955 at thirty-four weeks. Birth weight—4 pounds, 12 ounces—Female. Cord blood —Hb. 6.0 gm. per cent, Bil. 5.4 mgm. per cent. Exchange transfusion at three and one-half, thirty and fifty-four hours. At fifteen months development normal.

Case 3.—Baby H. Mother had an Rh-positive transfusion in 1940. Previous pregnancies—1945, miscarriage; 1947, stillborn fetal hydrops. Present pregnancy—antibody albumen 64 to 128. Induction March 22, 1955—no progress. Cesarean section March 24, 1955, at thirty-five weeks. Birth weight 4 pounds 13 ounces—male. Cord blood—Hb. 11.5 gm. per cent. Bil. 5.7 mgm. per cent. Exchange transfusion at twenty minutes, thirty-four and fifty-five hours. Simple transfusion at eighteen days. At twelve months, development normal.

Case 4.—Baby K. Previous pregnancies—1949, premature, died at 5 days; 1950, miscarriage; 1951, normal; 1952, stillborn fetal hydrops. Present pregnancy—antibody saline 8. Albumen 8. Surgical induction May 31, 1955 at thirty-five weeks. Birth weight 6 pounds—male. Cord blood—Hb. 11.8 gm. per cent. Bil. 4.5 mgm. per cent. Exchange transfusions at one and one-half, forty, sixty-four and one hundred ten hrs. At eleven months development normal.

Case 5.—Baby D. Previous pregnancies—1942, miscarriage; 1943, icterus gravis; 1944, stillborn hydrops; 1946 and 1947, icterus gravis; 1950 and 1951, stillborn hydrops. Present pregnancy—antibody 32 albumen. Surgical induction July 23, 1955 at thirty-four weeks. Birth weight—4 pounds, 4 ounces—female. Cord blood —Hb. 5.6 gm. per cent. Bil. 5.8 mgm. per cent. Exchange transfusions at two and one-half, ninety-six, and one hundred twenty hrs. At nine months development normal.

Case 6.—Baby H. Previous pregnancies—1938 and 1944, normal; 1944 and 1945, miscarriages; 1946, anencephalic; 1947, stillborn hydrops; 1949, icterus gravis; 1950, stillborn hydrops. Present pregnancy—antibody 32 albumen. Surgical induction Dec. 1, 1955 at thirty-five weeks. Birth weight 4 pounds, 13½ ounces—male. Cord blood—Hb. 10.3 gm. per cent. Bil 5.9 mgm. per cent. Exchange transfusion at three hours. Apparently normal at one year.

Case 7.—Baby N. Previous pregnancies—1938, miscarriage; 1940 and 1941, normal; 1944, icterus gravis died at 3 days; 1946 and 1951, stillborn hydrops. Present pregnancy—antibody 1 saline, 64 albumen. Surgical induction Jan. 6, 1956, at thirty-four weeks. Birth weight 4 pounds, 8 ounces—female. Cord blood—Hb. 10.2 gm. per cent. Bil. 5.4 mgm. per cent. Exchange transfusion at one and one-half and seventy-two hours. Apparently normal on discharge from hospital at twenty-one days.

Case 8.—Baby L. Previous pregnancies—1951, miscarriage; 1954, icterus gravis; 1955, stillborn hydrops. Present pregnancy—antibody 4 saline—16 albumen,—rose to 8 saline and 64 albumen. Surgical induction Jan. 15, 1956, at thirty-five weeks. Birth weight 4 pounds, 14 ounces—female. Cord blood—Hb. 7.0 gm. per cent. Bil. 6.8 mgm. per cent. Exchange transfusion at one hour of age. Apparently normal on discharge from hospital at fourteen days.

Case 9.—Baby W. (Brother of Case 1) Previous pregnancies—1947, normal; 1948, 1950, 1951, stillborn babies; 1954, induction at thirty-two weeks, four exchange transfusions. Present pregnancy—antibody 8 albumen. Surgical induction January 17, 1956, at thirty-two weeks. Birth weight 4 pounds, 15½ ounces—male. Cord blood—Hb. 6.9 gm. per cent. Bil. 4.5 mgm. per cent. Exchange transfusions at ten minutes, forty-nine and seventy-three hours. At fifteen months development normal.

Case 10.—Previous pregnancies—1945, normal; 1946, died at four hours, cause unknown; 1947, normal; 1949, stillborn, macerated. Present pregnancy—antibody 64 albumen. Cesarean section June 20, 1956, at thirty-six weeks. Birth weight 6 pounds, 8 ounces—male. Cord blood—Hb. 3.7 gm. per cent. Bil. 4.73 mgm. per cent. Exchange transfusions at thirty minutes, thirty and sixty hours. At ten months, development normal.

Case 11.—Baby L. Previous pregnancies—1949, miscarriage; 1950, normal; 1952, Kernicterus, survived damaged; 1953, icterus gravis, died 4 days; 1955, stillborn macerated. Present pregnancy—antibody 64 albumen. Surgical induction June 22, 1956, at thirty-four weeks. Birth weight—5 pounds, 1½ ounces—female. Cord blood—Hb. 6.0 gm. per cent. Bil. 5.56 mgm. per cent. Exchange transfusions at one and one-half, twenty, thirty-six and forty-six hours. Apparently normal on discharge from hospital at fifteen days of age.

Case 12.—Baby D. Previous pregnancies—1951, normal; 1953, mild hemolytic disease, simple transfusion, normal; 1954, icterus gravis; 1955, stillborn hydrops. Cesarean section October 6, 1956, at thirty-four weeks. Birth weight 4 pounds, 14 ounces—female. Cord blood—Hb. 8.1 gm. per cent; Bil. 3.96 mgm. per cent. Exchange transfusion at two hours of age. Simple transfusion to correct anemia (Hb. 4.6 gms. per cent) at four weeks of age. Development normal at nine months.

Conclusion

It seems that delivery at or before thirty-five weeks, followed by prompt, and if necessary, repeated exchange transfusions, will save the lives of over 80 per cent of infants with hemolytic disease of the newborn when the mother has had one or more previous stillborn infants due to Rh sensitization.

Addendum

Since presentation of this paper, there have been five additional cases in Rh-negative mothers with homozygous Rh-positive husbands where there have been several preceding stillbirths due to Rh immunization.

In this group, there are two surviving normal children following very early induction. In two, induction at thirty-five weeks was not early enough and fetal hydrops resulted; in one, the infant died of complications of prematurity unrelated to the moderate degree of hemolytic disease which was present.

This brings the total to fourteen survivors out of seventeen cases and in no way vitiates the desirability of early delivery in these cases.

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Medical Progress

Radiation Hazards in Medical Practice

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IT IS THE purpose of this article to summarize the various radiation hazards likely to be encountered in medical practice. The information presented was derived from measurements by the authors together with results of similar measurements by others as noted in the references. Brief consideration is also given to radiation hazards encountered in dental radiography and in shoe-fitting fluoroscopes.

The radiation hazard associated with the utilization of x-ray equipment has long been a concern of the careful user. However, not until recent years have studies of the effects of exposure provided a reasonable basis for evaluation of radiation damage to biological systems. With the advent of atomic energy and its potential hazard to the population of the whole world, much publicity has been given to this matter. The result has been a growing concern about radiation hazards by everyone regardless of his occupation. The physician should be even more concerned and be cognizant of the extent of the various radiation hazards because of his responsibility for the health of the community. This is important from the point of view of his patients, as well as for his own safety and that of his associates.

Effects of ionizing radiation are manifested in many ways, depending upon the biological systems involved and factors governing the exposure. Within months after Roentgen's momentous discovery of x-rays in 1895, pioneers in roentgenology, such as Dodd, developed severe dermatitis and submitted to first attempts at skin grafting for control of the skin lesions.[1] Daniel in 1896 reported a case of epilation following an attempt to demonstrate a metallic foreign body in the skull.[2] Before the end of the year, many workers presented severe x-ray-induced reactions. One of Edison's assistants, Clarence Dally, became the first known victim of x-rays, dying from "x-ray cancer."[3] That radiations from radioactive materials produced many of these same effects was also documented during this new era in science. The death of Madame Curie, winner of the Nobel Prize in nuclear chemistry, has been attributed to the effects of radiation.

Many dentists have had the misfortune of developing cancerous lesions on their fingers resulting from holding of dental films during radiography. The increased incidence of leukemia among radiologists is well documented.[4,5] This, together with the report that children of radiologists have a higher incidence of abnormalities than the offspring of other physicians,[6] points to an insidious common denominator—radiation exposure.

The biological changes ascribed to radiation exposure are initiated by the absorption of radiant energy in the system under consideration. This radiation may interact with atoms of a biological system to produce ionization leading to disruption of molecular bonds and the formation of highly oxidative radicals. Because the maintenance and growth of biological systems are dependent upon a multitude of chemical reactions which must be maintained in delicate balance, the absorption of radiant energy leads to a change in this balance with ultimate modification and/or destruction of the systems.

Investigations have shown that the ultimate effect depends upon the dose delivered, the time involved in its delivery and the type and energy of the emissions. There is evidence in some cases that at least partial recovery from radiation effects is possible. However, in the case of genetic changes most investigators agree that the effects

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of radiation are cumulative and irreversible. Thus it is expected that a non-threshold response would be observed when measuring genetic mutations as a function of dose delivered. In contrast to this, certain other biological effects produced by radiation are elicited only after a certain dose has been delivered. Once this threshold has been exceeded a very rapid rise in biological effect is noted with only a small increase in the dose delivered. An example of this latter mode of responsiveness is mortality in animals exposed to total body radiation. Figure 1 illustrates the general form of the curves obtained when plotting the biological effects of radiation as a function of dose delivered for both threshold and non-threshold phenomena.

Dosage of Radiation

Units for Measurement of Dosage.—The roentgen, "r," is a unit of radiation exposure indicating the amount of x- or gamma radiation required to produce a specified quantity of ionization in air under a particular set of conditions. With the advent of the medical use of radioactive isotopes another unit, the roentgen-equivalent physical ("rep") was defined to include ionization resulting from alpha, beta, and other particles. This latter unit has since been superseded by the "rad" which is a measure of energy absorbed (ergs per gram). As defined, one roentgen of x-rays produces an absorption of 87 ergs per gram of air, or about 93 ergs per gram of tissue. The number of ergs absorbed per gram of tissue will vary with the source and energy of the radiation as well as the nature of the tissue itself as is shown in Table I. At photon energies of 1,000,000 volts (1 MEV) the energy absorbed per gram of tissue per roentgen exposure is approximately 92 ergs for muscle, 96 ergs for fat and 85 ergs for bone. Conversely, at photon energies of 50,000 volts (0.05 MEV) these figures become 90 for muscle, 50 for fat and 400 for bone. Other values may result from the use of equivalent roentgens from corpuscular sources. To account for these differences, a unit of energy absorption, the "rad," was promulgated.

TABLE I. ENERGY ABSORBED
Ergs per gram per Roentgen delivered

Photon Energy (Kv)	Muscle	Fat	Bone	Air
50,000	87	78	110	87
1,000	92	96	85	87
500	92	96	87	87
100	91	86	170	87
50	90	58	400	87

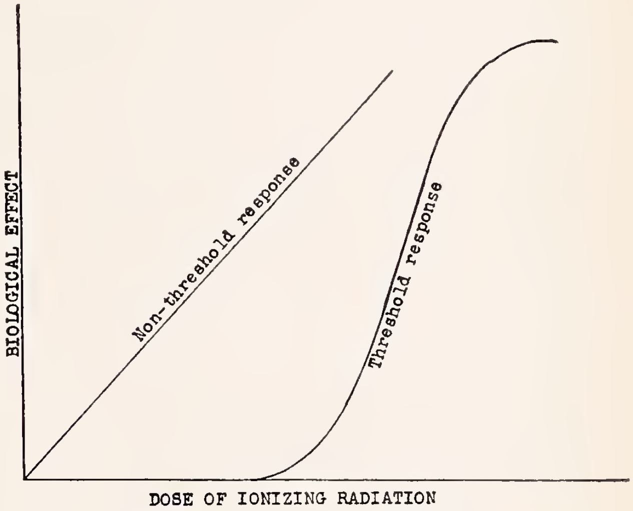

Fig. 1. Threshold vs. non-threshold phenomena.

Although biological effects depend upon the energy absorbed, this measurement in itself is not sufficient. The spatial distribution of the ionization produced is also important. Thus the effects of radiation on man are best measured in terms of the "rem" which is defined as the product of the "rads" delivered and the relative biological effectiveness ("RBE") of the radiation. From the foregoing one concludes that the effects of small

TABLE II. UNITS OF RADIATION DOSIMETRY

Unit	Equivalent
1 roentgen	Quantity of X or gamma radiation such that the associated corpuscular emission per 0.001293 grams of air, produces in air, ions carrying 1 e.s.u. of electrical charge of either sign 87 ergs/gram of air 93 ergs/gram of tissue
1 roentgen equivalent physical	Energy absorption dose in irradiated tissue of 93 ergs/gram
1 rad	Energy absorption dose of 100 ergs/gram
1 rem	1 Rad x RBE (relative biological effectiveness)
1 milliroentgen	.001 roentgen
1 millirep	.001 rep
1 millirad	.001 rad
1 millirem	.001 rem

doses of radiation are difficult to assess, and the quantitation of the dose delivered is also a major problem. A summary of these units of measurement is shown in Table II.

Consideration of Permissible Dose.—Since Müller's experiments with Drosophila thirty years ago, it has been known that ionizing radiations increase the gene mutation rate. The increase in the number of medical x-ray procedures and increased population exposure due to the development of nuclear power and weapons, has provoked much thought about the problem of "radio-genetics." It has been found, for example, that genes of mice are about fifteen times more sensitive to radiation-induced mutations than are those in Drosophila. The doses involved are well below those likely to produce observable somatic changes so that genetic considerations appear to govern the maximum permissible dose (MPD) to a population.

A base line for assay of harmful effects of small doses of radiation on a population can be established from background radiation. Sources of background radiation include cosmic rays and natural radioactivity in the earth, in building materials and in our bodies. Fallout from atomic weapons testing and contamination from the use of radioactive materials increases the level of exposure. The average exposure per person from natural background radiation is considered to be of the order of 3 millirems per week which would yield a cumulative dose of about 5 rems per generation (from conception to age thirty years). Some investigators have set this background level as high as 8 millirems per week particularly in Sweden where building materials have been found to contain a high concentration of radioactive material.[7]

There exists the postulate that spontaneous mutations are closely linked with background radiation.[8] The mutations occuring in germ cells of the gonads are considered to be the most important because of the involvement of future generations. Furthermore, all mutations appear to be deleterious as observed in experiments with fruit flies, various animals as well as results of accidental exposure of human beings. It is anticipated that the most frequent mutations will cause minor impairments of body function rather than gross changes. These effects will include increased susceptibility to disease, lower life expectancy and reduced fertility.

Evidence of an increase in the mutation rate in humans following radiation exposure may be drawn from the atomic bombings of Hiroshima and Nagasaki (although these data are not conclusive) and the results of a study by Macht and Lawrence that children of radiologists have a higher incidence of abnormalities than others.[6] Studies of progeny of parents who have received significant doses of pelvic irradiation also support the supposition that irradiation increases mutations in human germ cells.[9,10]

TABLE III. BACKGROUND RADIATION, MUTATION DOUBLING DOSE AND MAXIMUM PERMISSIBLE DOSE

Radiation	Dose
Average background	3 millrems/week 5 rem/generation
Mutation doubling dose	30-80 roentgens (or rems) 50 roentgens average
Maximum permissible dose	OCCUPATIONAL EXPOSURE 0.1 rem/week (recommended maximum for new installations) 0.3 rem/week 3.0 rem total in 13-week period 5.0 rem total in one year GENERAL POPULATION EXPOSURE Multiply factors above by 0.1 14,000,000 rems/1,000,000 population (to age 30)

In the light of genetic evidence available, it may be concluded that there appears to be no safe dose of irradiation. *Thus it becomes necessary to balance the genetic risk against the benefit derived from various medical procedures and nuclear development programs employing ionizing radiations.* The National Academy of Science has estimated that 30-80 roentgens constitutes a "doubling dose," that is, that this dose will double the spontaneous mutation rate.[11] On this basis, this group has recommended an MPD of 10 r. to the gonads during the period from conception to age thirty years. If 50 r, is then the average doubling dose, a population receiving an average of 10 roentgens will show a 20 per cent increase in genetic damage. This represents an increase in abnormalities in offspring attributable to genetic mutations from about 2 per cent to 2.4 per cent.[12] The above estimates have been based primarily on data derived from experiments with mice. However, data compiled from the atomic blasts in Japan indicate that mutation rates of genes in humans appear to be close to those observed in mice. From these and other considerations, the National Committee on Radiation Protection (NCRP) recommends that the MPD of 0.3 rem/week accepted prior to February, 1957,[13] be reduced so that individuals exposed to ionizing radiations because of occupation receive no more than 5 rems/year on the average. (These individuals may receive as much as 0.3 rem in any week, however, providing that no more than 3 rems are accumulated over a thirteen-week period.) For the population at large, a factor of ten lower in exposure is recommended which would yield an average annual dose of 0.5 rem. This latter recommendation is published in terms of a gonadal dose to the whole population not to exceed 14,000,000 rems per 1,000,000 people from conception to puberty which would average out to approximately 0.5 rem per year. A summary of these values is shown in Table III.

Sources of Radiation

Radiation exposure of an individual may originate from both external and internal sources. In most instances, exposure from external sources (principally x- and gamma rays) constitutes the greater hazard. Radioactive materials may serve as both internal and external sources of radiation.

TABLE IV. FACTORS DETERMINING HAZARD FROM RADIOISOTOPES

1. Quantity of material used
2. Body retention
3. Radiosensitivity of the involved tissues
4. Relationship of involved tissues (and organs) to body function
5. Effective half life of the isotope
6. Energy and character of the emanations

Radioisotopes when contained within the body present greater hazards than when they are limited to external sources because of the intimate contact and continuous irradiation of tissues surrounding the radioactive emitters. Furthermore, for some materials of long life time, there is essentially permanent deposition, such as, strontium^{90} or radium^{226} in bone.

Radioisotopes.—Except for radium and its decay products, there was little problem of radioactive contamination in the medical environment until the era of high voltage accelerators, nuclear reactors and atomic bombs. From the standpoint of medical usage there is an increasing number of isotopes which may present a hazard if the quantity is appreciable. Among these are I^{131}, P^{32}, Co^{60}, Fe^{59}, Au^{198}, and Cr^{51}. In weapons testing, possible contamination from appreciable quantities of Sr^{90} and Cs^{137} form the major hazard. Factors that determine the hazard of radioisotopes are listed in Table IV.

Some isotopes, such as Sr^{90}, I^{131}, and Fe^{59}, are particularly dangerous because they are readily metabolized and tend toward selective and prolonged localization. When one couples this problem with that of life time and the disintegration patterns, it is possible to assess in some measure the actual hazards involved. National Bureau of Standards Handbook 52 lists the radioactive isotopes together with maximum permissible concentrations (MPC) permitted in the body, air, and water based on the hazard considerations mentioned above. The MPC on the basis of the reduced MPD[13] in the body for some of these radioisotopes are shown in Table V.

TABLE V. MAXIMUM PERMISSIBLE CONCENTRATIONS OF SOME RADIOISOTOPES IN THE BODY

Element	Emission	Site of Localization	Effective Half Life (days)	MPC (uc)
Ra^{226}	alpha	bone	1.6 x 10^{4}	0.03
U (natural)	alpha	bone	30-120	0.003
Au^{198}	beta, gamma	kidneys	2.69	3.3
I^{131}	beta, gamma	thyroid	7	0.1
Sr^{90}	beta	bone	2.7 x 10^{3}	0.3
Co^{60}	beta, gamma	liver	9	1.0
Fe^{59}	beta, gamma	blood	27	330
Ca^{45}	beta	bone	151	22
S^{35}	beta	skin	18	33
P^{32}	beta	bone	14	3.3
Na^{24}	beta	total body	0.61	5
C^{14}	beta	total body	180	250

Roentgen Rays for Diagnostic Purposes.—The hazards associated with the use of x-rays for diagnostic purposes may be considered in three major categories: (1) the equipment, (2) protective devices, (3) safety habits.

Tables VI, VII and VIII summarize recommendations for control of hazard in fluoroscopy and radiography. This information was derived primarily from the specifications of the NCRP listed in National Bureau of Standards Handbook 60 on "X-ray Protection."

Hazard to the Operator of X-ray Equipment.—Fluoroscopy presents the greatest potential radiation hazard among the various diagnostic procedures using x-rays because of the time that may be involved. If one observes the precautions listed above, it is possible to control the radiation exposure to the fluoroscopist and assistants well below present MPD levels even for heavy schedules of work. The use of old machines that do not adhere to the specifications as listed by the NCRP may be a real source of difficulty. Adequate shielding in the tube housing and cone, shutters that operate properly, and sufficient filtration are all very important in eliminating unnecessary radiation exposure. The importance of using lead aprons, gloves and proper protective barriers cannot be overemphasized. A means for periodic radiation monitoring is also recommended. This can be done simply and effectively without great expense using dental film, special monitoring film or pocket dosimeters.

TABLE VI. FACTORS FOR CONTROL OF RADIATION EXPOSURE IN FLUOROSCOPY

Fluoroscope
1. Maximum of 0.1 r/hr./meter leakage radiation
2. Cone and adjustable diaphragm to limit the beam
3. 2½ mm. aluminum filter permanently fixed
4. Target-to-table distance at least 18 inches
5. "High-low" milliamperage change over switch
6. Cumulative timing device
7. 1.5 mm. lead equivalent material in fluorescent screen
8. 10 r/min. maximum dose at the table top
9. ¼ mm. lead equivalent drape during horizontal use

Protective Devices
1. 1.5 mm. lead equivalent in doors and walls to 7 feet
2. Leaded aprons and gloves worn by fluoroscopist
3. Radiation monitoring with film badges or pocket dosimeters
4. Leaded drapes overlying patient's gonads (when possible)

Safetv Habits
1. Trained personnel
2. Maximum utilization of inverse square law
3. Small field size and limit time of operation
4. Adequate dark adaptation
5. No holding of patients
6. Fluoroscopist's hands (with gloves) not placed in direct beam

Actually, good safety habits are the most important factor in controlling radiation exposure. Protection by distance (inverse square law), limitation of the field size, control of time, adequate dark adaptation, and avoiding the primary beam are all practices readily available to the careful fluoroscopist.

Scattered radiation through the bucky slot and from the patient and table top leads to significantly higher dose rates at the position occupied by the fluoroscopist during horizontal fluoroscopy than during vertical fluoroscopy. Therefore an additional leaded drape is recommended for use in horizontal fluoroscopy (Table VI).

The effect of field size and filtration on the radiation levels at various points of interest during fluoroscopy are shown in Figure 2.

In conventional radiography carefully executed, there is little risk of radiation exposure to the operator. Special techniques, such as urography, angiocardiography, aortography, and cerebral angiography requiring the presence of a physician and assistants in the radiographic room, produces a potential radiation problem which can be controlled by use of leaded drapes properly placed, in addition to maintaining the greatest possible distance from the x-ray beam. A number of reports illustrate the marked decrease in exposure to x-ray personnel that may be effected by simple safety considerations. For example, Ritvo and his associates reported that with use of proper coning, filtration and position, it is possible to reduce the dose to the physician's hands in urethrography from 66 mr. to less than 13 mr. per exposure.[14] Similar improvements have been found in angiocardiography. In femoral arteriography and lumbar aortography, use of a lead apron as a drape can reduce the exposure from 300 mr. to less than 20 mr.

Photofluorography deserves special mention because of its use in extensive surveys for tuberculosis and certain hospital admission procedures. Many of the older units were notoriously hazardous for the operators. The majority of the newer units have incorporated protective barriers and remotely located switches for controlling exposures. Studies of this hazard have been published by several authors.[15,16]

TABLE VII. FACTORS FOR CONTROL OF RADIATION EXPOSURE IN RADIOLOGY

Radiographic Machine
1. Maximum of 0.1 r/hr./meter leakage radiation
2. Cones or diaphragms to limit field size
3. 2½ mm. aluminum filter in medical units
4 1½ mm. aluminum filter in dental units
5. Equivalent filtration of table top less than 1 mm. aluminum
6. Exposure meter to limit time
7. Remote control switch operated from protected area

Protection Devices
1. 1.5-3 mm. lead equivalent in doors and walls to 7 feet
2. Radiation monitoring recommended
3. Leaded drapes overlying patient's gonads (when possible)

Safety Habits
1. Trained personnel
2. Maximum utilization of inverse square law
3. No holding of patients
4. Use of lead drapes if patient attendance is necessary
5. Limit number of exposures by careful technique

TABLE VIII. FACTORS FOR CONTROL OF RADIATION EXPOSURE FROM PORTABLE UNITS

1. Radiation monitoring is recommended
2. Trained personnel
3. No holding of patients or film cassette
4. Use of leaded aprons and drapes for patient and operators
5. Rotation of operators among the various x-ray diagnostic units
6. These units should not be used for fluoroscopy

Because of the confined areas for the operation of these units, great care must be exercised in the location of the x-ray personnel. Small changes in location can result in large differences in exposure. For this reason it has been recommended that a protection survey be made for all of these units.[15] Furthermore, it is recommended that personnel be rotated among the various tasks assigned in this survey program in order to keep the exposures to any one group below the MPD.

Another diagnostic procedure involving unusual hazards to the operator is dental radiography, in which exposures to the dentist may be as high as 1.5 roentgens per hour of operation.[17] The practice of holding the film in the patient's mouth must be prohibited for reasons already mentioned. The operator of a dental x-ray machine may receive a total body dose of 125 milliroentgens per full mouth set of x-rays if care is not exercised.

Shoe-fitting fluoroscopes, usually operated at 50 KV, 3-8 MA, 7.5-20 cm. FSD and with 0.1 mm. aluminum filter, are considered hazardous unless the operator is very radiation-protection conscious. Scattered radiation in the excess of 500 milliroentgens per hour has been measured at the position normally occupied by the shoe salesman. Thus the annual MPD might be received in ten hours of operation. If each fitting with such a unit is limited to four exposures each of five seconds in duration as has been recommended,[18] a total of 1,800 fittings might be made annually by an operator (on the basis of 5 roentgens/year as the MPD). This averages to approximately six fittings per day. It is expected that in some cases more than four exposures are made per customer which would further decrease the number of actual fittings per year that are permissible from the radiation hazard standpoint.

Patient Hazards from Radiation

Even as fluoroscopes present the major hazard to the operator, so these units also constitute the greatest potential danger to the patient.* A dose rate of 10 roentgens per minute is permitted at the table top of a fluoroscope; therefore long periods of exposure will result in a sizable patient dose. In a radiation hazard survey of fluoroscopes with no filtration, and with a short focal spot to table top distance, we have measured dose rates in excess of 35 r./min. at table top. Under these conditions, the patient may very well receive a dose sufficient to produce a sharp erythema. Bell has referred to the patient hazard during fluoroscopy in an article appropriately entitled "X-ray Therapy in Fluoroscopy."[19] He reported that under extreme conditions, as during gastrointestinal fluoroscopy at 80 KVP, 3 MA, and no added filter that a patient may receive a skin dose of 400 r. and a dose of 47.5 r. at a depth of 10 cm. in the tissues. A summary of measurements of patient exposure under varying conditions of fluoroscopy are shown in Table IX. These measurements serve to emphasize the importance of filtration, control of time and the limiting of field size to keep the integral dose as low as possible.

FLUOROSCOPE (90 KV, 3 MA)

Position	Filtration 1 mm. Al		Filtration 3 mm. Al	
	7 x 9 *	4 x 4 *	7 x 9 *	4 x 4 *
A	12 r/min.	12 r/min.	7.2 r/min.	7.2 r/min.
B	0.38 r/min.		0.35 r/min.	
C	4.2 mr/hr.	4.2 mr/hr.	4.0 mr/hr.	4.0 mr/hr.
D	550 mr/hr.	180 mr/hr.	400 mr/hr.	90 mr/hr.
E	240 mr/hr.	50 mr/hr.	200 mr/hr.	45 mr/hr.
F	450 mr/hr.	110 mr/hr.	300 mr/hr.	70 mr/hr.
G	20 mr/hr.	15 mr/hr.	15 mr/hr.	12 mr/hr.
H	40 mr/hr.	19 mr/hr.	30 mr/hr.	17 mr/hr.
I	6 mr/hr.	6 mr/hr.	6 mr/hr.	6 mr/hr.

* Field Size (inches)

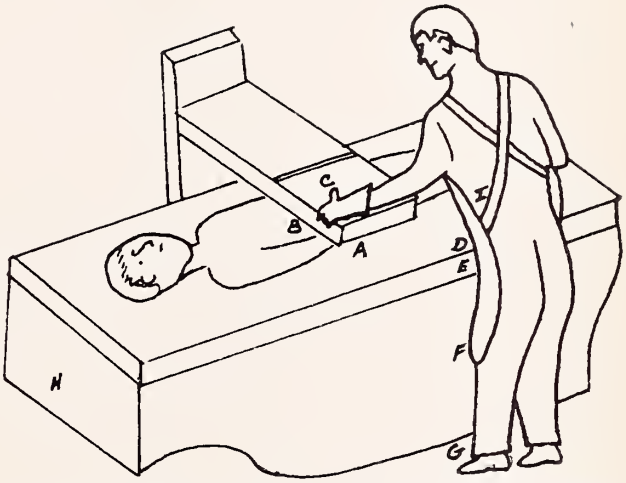

Fig. 2. Effects of field size and filtration on radiation exposure in fluoroscopy.

Patient exposure from radiographic installations may reach hazardous levels if the filtration of the machine is inadequate, if the primary beam is not restricted by coning, and if the number of radiographs is not carefully controlled. Average exposures for conventional radiographic techniques using x-rays filtered by 2 mm. of aluminum and with field size limited by cones or diaphragms are summarized in Table X. These doses represent an average of our measurements together with those reported by others.[20-23]

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*The use of fluoroscopy for direct visual reduction of fractures or for foreign body removal is extremely hazardous for both patient and physician. There are many cases of skin malignancy on the hands of physicians who have used this technique. Indeed, rarely, if ever, is there indication for this type of fluoroscopy. For reduction of fractures and foreign body removal, radiographic techniques are far safer for all concerned and furthermore, provide a permanent record.

TABLE IX. PATIENT EXPOSURE IN FLUOROSCOPY

	Machine 1	Machine 2	Machine 3
Exposure (table top)	36 r/min.	6 r/min.	1.5 r/min.
Skin dose (5 min.)	180 r	30 r	7.5 r
Integral dose (5 min.) 10x10 cm. field	52,000 gram-r (560 ergs)	17,300 gram-r (186 ergs)	4,320 gram-r (47 ergs)
20x20 cm. field	208,000 gram-r (2240 ergs)	69,000 gram-r (743 ergs)	17,250 gram-r (185 ergs)

Machine 1—90 Kv, 4 MA, no filter, FSD 15 inches.
Machine 2—90 Kv, 3 MA, 2½ mm. Aluminum, FSD 18 inches.
Machine 3—Image amplifier with machine 2.

It may be seen that the skin dose to a patient's chest is considerably higher in photofluorography than for conventional 14 x 17 inch radiographs of the chest. The average skin dose per exposure for the former was found to be about 750 milliroentgens whereas with the 14 x 17 inch plate the average dose was found to be 30 milliroentgens. This represents about a twenty-five-fold difference in exposure. A corresponding difference in the gonadal dose would be anticipated and has been confirmed in the measurements reported by Webster and his associates.[20]

For exposures in which the gonadal dose varies appreciably with sex, both values are given. It is noteworthy that the use of a leaded apron to protect the gonads for x-ray procedures not involving this region permits a reduction of the gonadal dose by a factor of about four.[24]

In considering the radiation hazard to the shoe buyer from shoe fluoroscopes, it is worthy of note that recently an ordinance was passed by the city of Minneapolis forbidding the use of these units. Williams has reported exposures of 10-116 roentgens to the feet during the fitting of shoes.[25] The upper limit appears particularly high if the fluoroscope is properly filtered and the exposure time is limited. We have surveyed units in which the dose to the feet during a five-second exposure has varied from 0.67 to 6 roentgens. The top figure represents a unit without sufficient filtration and when 1 mm. aluminum was added to the unit, the dose delivered in five seconds was reduced to less than 1.25 r.

The gonadal dose per exposure will vary considerably depending upon the coning of the primary beam and the protective barriers incorporated into the shoe fluoroscope. If these factors are not controlled, the gonadal dose per five-second exposure can exceed 20 mr. Thirty such exposures would constitute the annual MPD for this individual indicating that unless care is exercised, shoe fitting fluoroscopes may be termed a definite health hazard.

Radiation therapy also carries a somatic and genetic risk for the patient. When treating malignant disease, there can be no question that the risk is justified. However, the use of x-rays and radium in treating benign conditions, particularly those of the skin, such as acne, neurodermatitis, hemangiomata, and verrucae of the hands and feet, must be carefully limited to those conditions which cannot be effectively controlled by other methods.

TABLE X. PATIENT EXPOSURE IN RADIOGRAPHY
Average exposure (milliroentgens) for conventional techniques using x-rays filtered by 2 mm. of aluminum and with field size limited by cones and diaphragms

Part of Anatomy	View	Skin Dose (mr.)	Gonadal Dose (mr.)
Skull	AP	600	.02
Shoulder	AP	200	.02
Hand		120	.03
Chest	PA	30	.03
Chest (P. R.)*	PA	750	.75
Abdomen	AP	550	20 (150)**
G. I. Series	PA	900	5 (50)
	Lateral	2000	10 (60)
Barium enema	PA	1000	30 (200)
	Lateral	2500	40 (270)
Spine (lumbar)	AP	800	15 (150)
	Lateral	2300	40 (240)
Pelvis	AP	600	450 (150)
	Lateral	2000	1500 (400)
Knee	AP	40	0.3
Foot	AP	20	0.2

*Photofluorogram.
**Indicates gonadal dose to females when significantly different from males.

Radioactive isotopes for most clinical purposes carry practically no radiation risk, except in the presence of pregnancy. We feel that it is desirable to withhold even small tracer doses of radioisotopes in pregnancy because of potential hazards to the fetus. There is some debate as to the relative radiation hazard when using radioactive iodine to treat hyperthyroidism in patients under thirty-five years of age; the hazard of inducing thyroid malignancy is as yet theoretical and must be balanced in the physician's evaluation against the known small, but nevertheless real, hazards of other therapeutic methods. It is unlikely that other properly conceived human uses of radioisotopes will represent any real hazard to patients.

Discussion

How might one determine his exposure to radiation over a period of months and years? For the individuals whose occupations require the use of ionizing radiations, this problem is most efficiently handled by the use of film monitoring badges or pocket dosimeters carried at various parts of the body. The exposures received can be logged for a continuous record. Many hospitals are doing this routinely to safeguard the health of their workers.

For the population in general, this task is much more difficult if not impossible to carry out satisfactorily. The National Committee on Radiation Protection has given thought to this problem to assist the state health departments in setting up specifications for the control of radiation hazards.[26] Consideration has been given to a radiation diary to be carried by everyone from the cradle to the grave; however, the problems in administering such a program are almost overwhelming to say nothing about the added instrumentation and training required to make logical estimates of gonadal dose for all exposures. For example, there are some 100,000 diagnostic x-ray units in operation in the United States with only about 5,000 certified radiologists. Even among this group of specialists, there would be considerable difficulty in estimating gonadal doses for all exposures.

Summary

All of us should be aware that ionizing radiations may constitute real health hazards. This does not mean that we should bar the doors of all x-ray departments and remove all radioisotopes from medical use. One cannot question that when used properly, ionizing radiations can be an asset to better health rather than a liability. The use of x-rays aids in the diagnosis of many patient disorders, such as tuberculosis, disfunction of internal organs, tumor growth, fractured bones, and dental disease to mention just a few. Radioactive isotopes are being used more and more for such diagnoses as thyroid function (I^{131}), blood volume (Cr^{51}, RISA), red blood cell survival (Cr^{51}), iron metabolism (Fe^{59}), pernicious anemia (Co^{60}-Vitamin B_{12}), tumor detection (P^{32}), fluid balance (Na^{24}), and metabolism of tissues (P^{32}, Na^{24}, Ca^{45}, C^{14}). The utilization of atomic energy for production of power may improve our economy and lead to an even better standard of living than we now enjoy.

TABLE XI. PATIENT EXPOSURE FROM DENTAL X-RAY UNITS

Doses to the skin: 65 KVP, 10 MA, 3 seconds

Machine	No Filter	With Added Filter	With Added Filter and Fast Film
1	1.0 r	(½ mm. Aluminum) 0.66 r	0.22 r
2	2.8 r	(2½ mm. Aluminum) 0.7 r	0.24 r
3	2.4 r	(1mm. Aluminum) 1.4 r	0.5 r
4	2.4 r	(1 mm. Aluminum) 1.3 r	0.33 r
5	4.0 r	(2½ mm. Aluminum) 2.2 r	0.7 r
Maximum estimated dose to skin for 14 exposures (full mouth series)			
1	9 r	6 r	2 r
2	26 r	6.5 r	2.2 r
3	22 r	13 r	4.7 r
4	22 r	12 r	3.1 r
5	37 r	20 r	6.5 r
Gonadal dose estimated per full mouth series			
	4-5 mr	.2 mr.	less than 1 mr.

All machines properly coned. Approx. FFD 14 inches.

When utilizing ionizing radiations one should employ the best protection possible. Unnecessary fluoroscopic or radiographic examination or unnecessary use of radioisotopes for diagnostic purposes should not be undertaken any more lightly than the unwarranted use of hazardous drugs. The use of ionizing radiations for diagnostic purposes should not detract in any way from complete patient histories and careful physical examinations.

Patients in the childbearing age group should be exposed as little as possible to radiations from x-rays and radioactive isotopes. Oftentimes during pregnancy, it is desirous to eliminate completely the use of radioisotopes and to sharply curtail the use of x-rays except in unusual cases. In these special cases leaded drapes overlying the patient's gonads should be employed when possible.

In fluoroscopy and radiography the field size should be limited to the area of interest. This is particularly true when it is necessary to use x-rays for examination of infants who might otherwise receive radiation to the total body. Here again leaded drapes for gonadal protection should be used if the examination permits.

Training in the proper use of ionizing radiations as a diagnostic tool cannot be overemphasized. Adequate filtration of the x-ray beam and protection devices for the patient, doctor, and technicians are important factors in minimizing the radiation hazards from x-ray machines and radioisotopes. High kilovoltage techniques, high speed film and intensifying screens consistent with the diagnostic problem are recommended.

An awareness of the attenuation of radiation by distance (inverse square law) together with other safety habits listed in Tables VI, VII, and VIII illustrate other significant measures for reduction of radiation exposure.

Cognizance of radiation hazard coupled with good judgment and common sense[27] will go far toward reducing the exposure of our whole population to ionizing radiations for diagnostic purposes below that resulting from natural background. On this basis, it is our belief that no injustifiable genetic problems in future generations need be anticipated from diagnostic procedures involving the use of ionizing radiations.

In final analysis, *the physician is morally and ethically bound to expect realistically a beneficial result for the patient for a radiation method in order to justify any exposure, no matter how small in amount. Furthermore, the physician must extend every effort to keep this exposure at an irreducible minimum.*

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Case Presentation

The Overuse of Steroid Therapy in Bronchial Asthma

Report of Case of Iatrogenic Cushing's Syndrome Due to Corticotropin

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A RATHER new complication of asthma has arisen since the advent of steroid therapy. The term "steroid therapy" is used in a broad sense in this paper to include treatment of the patient with either corticotropin or cortisonelike steroids. The problem is the asthmatic patient who is dependent on exogenous steroids (or ACTH) for relief. These patients usually will not tolerate anything less than complete relief of their asthma, and they are not willing to endure periods of incomplete relief which reduction or stopping of the steroid therapy involves. As a result they continue steroid therapy indefinitely. Frequently patients are seen with complications which have arisen from the prolonged use of these potent drugs. Recently Kern[1] has pointed out the abusive use of steroid therapy in various fields of medicine, including allergy and especially the asthmatic patient. There is no question that ACTH and cortisone and some of its analogues are helpful in the treatment of asthma. In fact, at times they are lifesaving, but it should be remembered that they are not curative and that they provide symptomatic relief only. Their use, therefore, should be restricted to as short a period as possible so that the undesirable effects of these potent substances will be minimized.

The case being reported is a good example of prolonged and excessive use of ACTH that produced a typical exogenous Cushing's syndrome. The patient came to the clinic because he was tired of taking injections of ACTH and because hypertension, obesity and acne had developed. He was willing to stay in the hospital several weeks to see if he could discontinue the use of ACTH.

Report of Case

On registration at the Mayo Clinic the patient reported that asthma had developed six years previously, following an upper respiratory infection and bronchitis. The administration of ACTH had been started by his home physician for relief of severe asthma which followed the bronchitis and hard coughing. He had been a smoker for thirty years and was using about a pack of cigarettes a day. At one time in his life he had been a chronic alcoholic but had been able to stop drinking. There was no history of allergy in the family. The patient stated that when he took sulfa drugs a rash developed. In 1951 he was depressed and was given seventeen electroshock treatments by a psychiatrist. It is not known whether ACTH was responsible for any of his mental trouble. Subsequent to this he was able to work with a construction company but always with the aid of ACTH which he gave to himself; he regulated the dosage as he thought best and averaged about 20 units every 36 hours.

The patient was a pudgy white man, 55 years of age and 5 feet 8 inches tall; he weighed 185 pounds undressed. His blood pressure ranged from 155 to 170 mm. of mercury systolic and 110 mm. diastolic. His pulse ranged from 96 to 100 beats per minute. He had the features of marked hypercortisonism with moon facies, seborrhea, supraclavicular and cervicodorsal fat pads and an acneiform eruption over his back. He also had hyperkeratotic lesions over the left elbow with an underlying olecranon bursitis. There was evidence of tinea pedis as well as a fungous infection of the toenails. Examination of the eyes gave negative results except for narrowing of the retinal arterioles, grade 2, and hypertensive sclerosis, grade 1. On rectal examination it was found that he had a few internal hemorrhoids. The results of all skin sensitization tests were negative. Results of other laboratory studies, which included tests for serum sodium, serum potassium and blood urea; sputum for tests of acid-fast organisms and malignant cells and for general culture;

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determination of hemoglobin, sedimentation rate, plasma cholesterol, blood chlorides and erythrocytes; routine urinalysis, and serologic tests for syphilis, were reported negative or within normal limits. Roentgenograms of the lumbar portion of the spinal column and the sinuses did not show evidence of abnormality. The electrocardiogram showed a rate of 98 with sinus rhythm and changes compatible with hypertension. He had persistent leukocytosis with a cell count ranging from 13,200 to 19,000, a condition which has been observed in other patients treated for long periods with steroids.[2] A differential count showed 13.0 per cent lymphocytes, 0.5 per cent monocytes, 86.0 per cent neutrophils, and 0.5 per cent metamyelocytes. A blood smear showed neutrophilic leukocytosis but no toxicity or immaturity.

Twenty-four hours after the last injection of ACTH as an outpatient, the patient was hospitalized in the metabolic unit from June 3 to 29; repeated determinations of urinary 17-ketosteroids, corticosteroids,* and plasma corticosteroids[4] were made following the withdrawal of corticotropin, the intramuscular injection of 20 units of ACTH, and the intravenous injection of 0.1 unit of regular insulin per kilogram of body weight. On the days that the patient received ACTH, the hormone was administered immediately after blood was withdrawn at 8 a.m. for determination of plasma corticoids. Blood was withdrawn again at 11 a.m. and 3:30 p.m., three and seven and one half hours after injection of ACTH, except on Saturday, June 7. Insulin was administered immediately after blood was obtained at 8 a.m. for determination of plasma corticoids and blood was withdrawn again for plasma corticoid determinations at 9 and 9:30 a.m. and at 3:30 p.m., one hour, one and one half hours and seven and one half hours after injection of insulin. On control days blood was obtained at least twice a day for plasma corticoids (8 a.m. and 3:30 p.m.) and occasionally three times a day (8 and 11 a.m. and 3:30 p.m.) (Fig. 1).

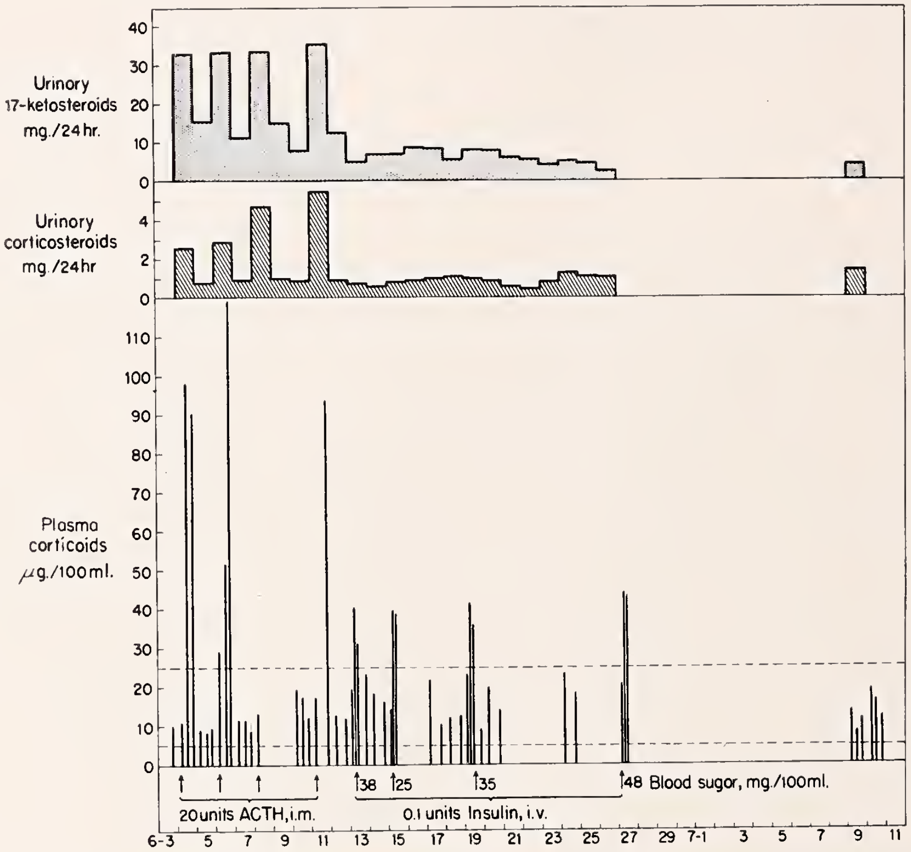

Fig. 1. Results of determinations for urinary 17-ketosteroids and corticosteroids and for plasma corticosteroids after injections of ACTH or insulin and after withdrawal of ACTH.

The response to the intramuscular injection of 20 units of ACTH, as indicated by values for urinary 17-ketosteroids, corticosteroids and plasma corticosteroids, was prompt and very intense indicating the absence of adrenal atrophy and suggesting that the adrenal glands were probably hyperplastic. A prompt and normal increase in the concentration of plasma corticosteroids followed insulin-induced hypoglycemia and indicated that the pituitary body was not atrophic in so far as its ability to release ACTH in response to hypoglycemia was concerned.

(Turn to Page 401)

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*Urinary corticosteroids were determined by a method similar to that of Corcoran and Page.[3]

From June 16 to 27 the patient's asthma gradually became worse, but the concentrations of corticosteroids in the urine and plasma were within normal limits. Thus, there was no evidence that the increase or aggravation of the asthma was due to adrenal cortical insufficiency. It could be concluded also that the function of the patient's pituitary and adrenal glands was not seriously impaired in spite of prolonged therapy with exogenous corticotropins.

Why then did the asthma become worse after cessation of ACTH treatment? One can speculate that removing the suppressive effect of the exogenous ACTH set the stage for local tissue swelling in the bronchial mucosa and caused a rebound effect, with a decrease in size of the lumen of the bronchi so that wheezing and coughing began. We know that cortisone and ACTH have powerful suppressing effects on bacterial and chemical inflammation as well as antiallergic and antirheumatic effects. It is conceivable then that discontinuation of these drugs after the tissues had become accustomed to them results in the recurrence of turgescence; and possibly that inflammation of a latent chronic infection, being no longer suppressed, again became a detrimental factor causing mucosal swelling also with resultant asthma.

It is of interest that on June 16, five days after the patient had received his last dose of ACTH, his asthma began to flare up. The next day vasomotor rhinitis developed and the rash became worse. At first it was thought that an iodide rash was developing but this was not substantiated. On June 18 he felt slowed down and tired. On June 19, it was noted that there was less bruising at the venipuncture sites and his face was not so red as at the beginning of the observation period. On June 20, his coughing and wheezing began to get much worse; requiring more and more antiasthmatic measures, such as intravenous and rectal administrations of aminophylline, amodrine, iodides, isuprel by nebulizer, elixophyllin orally and epinephrine subcutaneously, until he was utilizing about all that could be used.

TABLE I. PULMONARY FUNCTION TESTS

Age and Sex: 55 yr., M. S. A.: 1.95 sq. m.		Ht.: 175.3 cm. Wt.: 79.5 kg.	
		Test	
Pulmonary Volumes	Estimated Normal	June 3, 1957	June 27, 1957
Vital capacity (VC), liters	4.7	3.26 3.31*†	1.97 1.88*‡
Residual volume, liters	1.6	1.78	
Total capacity (TC), liters	6.3	5.04	
RV/TC x 100, per cent	40	35	
Functional residual capacity, liters	2.8	2.63	
Inspiratory capacity, liters	3.5	2.47	1.70
Expiratory reserve, liters	1.2	0.86	0.37
Maximal breathing capacity, liters per minute	114	91 84*†	36 34*‡
Expiratory slowing, grade	0	2	4
Air trapping	0	?	Present
Nitrogen washout index, per cent	2.5 or less	1.4	
Arterial oxygen saturation (oximeter)		Normal	Normal

*After administration of aerosolized bronchodilator.

†June 3, 1957: Exercise limitation is out of proportion to other findings. Some obstructive phenomena and probably reduced vital capacity and total capacities are abnormalities. At this time patient was on ACTH therapy.

‡June 27, 1957: Findings indicate remarkable reduction in capacity and increase in obstructive phenomena since last test. Patient had been off ACTH therapy for 2 weeks.

The results of tests for pulmonary function done on June 3 and 27 are shown in the table. The results obtained on June 27 were of interest because at this time the patient had not had any ACTH for about two weeks. The data show definite decrease in lung capacity as well as an increase in obstructive phenomena.

About this time it became apparent that steroid therapy would be required to control the severe asthma. It was thought that steroid therapy could be controlled more easily with an oral preparation of a steroid than with ACTH. Consequently, he was started on 40 mg. of prednisolone a day in an anticipated program of reduction which was designed for gradual but complete disuse of the drug by mid-October. A telephone call from the patient on July 15 revealed that he had lost 23 pounds, some of his facial swelling and about five and one half inches in his waistline, and that he was working at his old job.

Comment

This case demonstrates the new complication of asthma which we are seeing more frequently—a condition in which the patient is literally trapped with use of the steroids or ACTH. He cannot discontinue therapy without having a flare-up of asthma. The longer steroid therapy is used the more difficult discontinuation becomes, and more time is allowed for other complications to develop. The problem arises of how to go about stopping the patient's use of ACTH or steroids.

At present the only method appears to be that of gradually reducing the dosage in steps individualized to fit each patient[5] so that the asthma does not flare up. If it does the dose of ACTH or steroid may have to be increased temporarily before further reduction can be attempted. This reduction is not easy because of the likelihood of flare-up and the long time required to reach the objective. To achieve independence from the drugs it is necessary that the physician have the patient's complete co-operation and for the patient to know that the reduction program is going to take considerable time. The patient should be willing to undergo some symptoms of asthma without insisting on immediate or complete relief of wheezing. The dosages of steroids and ACTH should be given at regular intervals throughout the 24 hours. Patients exposed to added stress, such as, surgical measures or upper respiratory infections, should be supported with extra amounts of cortisone until the period of stress is past. For any contemplated operation it has been the custom at the clinic to give patients using steroids, or those who have had a considerable amount of steroids within the past several months, 200 mg. of cortisone intramuscularly 48, 24, and 2 hours before operation followed by tapering doses of cortisone for the next three to four days.

It is not advisable to allow asthmatic patients to get started on long-term treatment with steroids or ACTH. They should not be given these drugs unless absolutely necessary, that is, unless they are becoming more seriously ill and are not responding to the usual conservative measures. If steroid drugs must be given, they should be discontinued as soon as possible. As stated previously, they are not a cure; they provide only symptomatic relief. Short courses of ten days or less should not be harmful. Prolonged use may lead not only to dependence on the drug but also to other complications, such as, hypertension, osteoporosis, peptic ulcer, spreading of infection, flare-up of tuberculosis, development of diabetes, edema, and possible psychosis. The simpler antiasthmatic measures should always be tried first while the cause of asthma is being determined, for there is always a cause, if it can be found. Complete examinations of the ear, nose, and throat, including roentgenograms of the sinuses, are essential. Skin sensitization tests should be made and the patient should be instructed in avoidance of allergens to which he is sensitive or a program of hyposensitization should be outlined, if indicated. Respiratory irritants, such as smoke fumes or smoking, sudden temperature changes, dust, feathers, cold air, overcoughing and recurrent respiratory infections, should be avoided. Ideally steroids should not be used as the first form of therapy, although in status asthmaticus it may be necessary until a thorough examination can be done if other measures are not effective.

Summary

A case is reported in which a patient had typical exogenous Cushing's syndrome due to excessive use of corticotropin for control of asthma. A severe flare-up of asthma occurred after treatment with corticotropin was discontinued. The cause of flare-ups of asthma following discontinuance of corticotropin is not known; in this case there was no evidence of adrenal or pituitary suppression. It is known that corticotropin and cortisone-like steroids have powerful anti-inflammatory effects and when their use in patients with asthma is discontinued a "rebound phenomenon" due to local tissue swelling may follow. Prolonged treatment of asthmatic patients with corticotropin or steroids should be avoided whenever possible to prevent serious complications, among which is the problem of steroid dependence.

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DEHYDRATION FOR THE COMMON COLD

Dehydration for the common cold, a complete reversal of the commonly recommended procedure of forcing fluids, "usually produces spectacular results," reports Dr. Guy T. Vise of Medidian, Mississippi, in the *Southern Medical Journal* (December, 1957).

Several hundred patients with the common cold have been treated over the past five years by dehydration. Good or excellent results have been obtained in over 90 per cent of the cases, he added.

The routine developed by Dr. Vise consists of the limitation of fluids to 1000 cc. daily—one cupful of soup, fruit juice, vegetable juice, tea, coffee or a fruit every three hours while awake—no water is permitted and very little or no salt. Other measures include a saline laxative, antibiotics every four hours, aspirin, codeine, antihistamines and a hot sweat pack for 2 hours once daily.

The average loss in weight that produces effective symptomatic results is 2 to 4 pounds for an adult or roughly 2 to 3 per cent of the body weight.

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CASE PRESENTATION—MINNEAPOLIS VA HOSPITAL

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DONALD F. GLEASON, M.D.
HOWARD F. HORNS, M.D.

This was the first and only admission to our hospital for this fifty-nine-year-old white married man from West Concord, Minnesota. His previous occupations had included farming, road construction and management of a tavern. He came to the hospital because of listlessness, easy fatigability and intermittent epigastric pain for the previous nine months. The pain was a constant ache but varied in severity, occasionally being somewhat relieved by eating small meals. It was aggravated by a large meal but was relieved by the ingestion of copious quantities of beer or whiskey. Approximately one month prior to admission, the patient experienced a sudden attack of severe pain in the right upper quadrant of the abdomen, which persisted for an entire day. It was not associated with chills or with a fever. Following this his urine appeared darker for several days. He noted dark urine on several occasions subsequently, but no jaundice. He had been extremely ill with jaundice in 1921.

The gastrointestinal history was otherwise negative. Specificallly, there was no history of emesis, hematemesis, melena or change in bowel habits.

The past history revealed that he had developed a Bell's Palsy on the left side of his face in 1941. A residual weakness of the left facial musculature still remained, the patient retaining food within the left buccal area and being unable to prevent his eye from closing during deglutition. Tinnitus had been present in the left ear for the two years prior to admission. In 1940 he had sustained bilateral lacerations of the face in an automobile accident. He had had an appendectomy and repair of a scrotal hernia in 1914. His alcoholic intake was considerable, amounting to a pint of whiskey daily in some previous years. He was a snuff chewer but had given up smoking in 1945. Epistaxes from the right side of his nose had troubled him since childhood. The family history revealed that his father had died of leukemia. The past history, family history and system review were otherwise negative.

On physical examination the patient appeared as a well developed, plethoric and obese person—not acutely ill. The temperature was 98.6 degrees F., the pulse rate 72 and regular, and the blood pressure was 152/104. He weighed 192 pounds and was 5 feet 8 inches tall. Several firm, non-tender lymph nodes were easily palpated along the cervical chains bilaterally and were also felt in the left axilla. The left upper eyelid was ptotic. A cataract was present in the left lens. The liver was very large, firm, tender and showed possibly some nodularity below the edge on the right. The spleen could not be palpated. The scars of a previous appendectomy and right inguinal herniorraphy were present and were well healed. The prostate was enlarged but felt benign. The physical examination was otherwise within normal limits.

Laboratory tests revealed that the hemoglobin was 17.8 gm. per cent and the leukocyte count was 9,500 with a differential count of 64 per cent neutrophils, 32 per cent lymphocytes, 3 per cent eosinophils and 1 per cent basophils. The reticulocyte count was .6 per cent. Two urinalyses were normal. The erythrocyte sedimentation rate was 6 mm. in one hour, Westergren method. The clotting time was sixteen minutes and the bleeding time was four minutes. The platelet count ranged from 313,000 to 163,000 on four determinations. The VDRL slide test was negative. The blood urea nitrogen was successively 10 and 14 mg. per cent. The fasting blood sugar was 87 mg. per cent. The serum sodium was 139.0 and the potassium 4.7 milliequivalents per litre. The serum calcium was 10.5 mg. per cent and the serum phosphorous 4.1 mg. per cent. The serum acid phosphatase was 1 KAU and the alkaline phosphatase was 23 KAU. The serum albumin was 4.6 and the globulin 1.7 gm. per cent. The serum bilirubin was 1.1 mg. per cent. At the same time the bromsulphalein retention was 24.2 per cent after forty-five minutes, in the absence of fever. The thymol turbidity was 0.4 units and the cephalin flocculation was negative. Direct and indirect Coombs' tests were negative. A qualitative test for urinary porphobilinogen, using Ehrlich's aldehyde reagent, was negative, but a trace of color was absorbed into the chloroform layer. Two random stools, collected five days apart, contained 3.8 and 96.2 Ehrlich units per cent of urobilinogen. Another stool was negative for blood.

A roentgenogram of the chest revealed elevation of the right diaphragm, but was otherwise normal. A film of the abdomen showed enlargement of the liver and spleen. An intravenous pyelogram and barium studies of the upper and lower gastrointestinal tract were normal. Two cholecystograms revealed no filling of the gall bladder, even after a double dose of dye. A biopsy of an axillary lymph node was interpreted as showing chronic lymphocytic leukemia and a sternal marrow biopsy was interpreted as being compatible with this diagnosis.

The patient remained ambulatory and afebrile until his fourth hospital day, when he complained of an increase in pain in his right upper quadrant. He had severe shaking chills, nausea and vomiting. His temperature was 101 degrees F. The leukocyte count had now decreased to 2500 with a differential count of 45 per cent neutrophils, 53 per cent lymphocytes and 2 per cent monocytes. Two blood cultures grew out no organisms, but he was treated with penicillin and oxytetracycline. He promptly improved but continued to complain at

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times of pain in the right upper abdomen, associated now with right shoulder-strap pain. His spleen became palpable. Following a course of nitrogen mustard, he felt better and on his twenty-ninth hospital day was allowed to return home for two weeks.

When he returned, however, he felt weaker and more fatigued. He was now febrile and seemed icteric. His serum alkaline phosphatase was now 35.5 KAU and his total serum bilirubin had risen to 2.1 mg. per cent. The leukocyte count was 3100 with a differential count of 56 per cent neutrophils, 37 per cent lymphocytes, 5 per cent monocytes, 1 per cent eosinophils and 1 per cent basophils. The hemoglobin had decreased to 13.4 gm. per cent and the reticulocyte count was 2.1 per cent. The erythrocyte sedimentation rate had risen to 59 mm. in one hour. Fifty-six days after his initial admission to the hospital, the prothrombin activity was 66 per cent. Because it was felt that his deepening jaundice might be due to compression of the common bile duct by enlarged leukemic lymph nodes, x-ray therapy to the area of the porta hepatis was begun three days later and continued for two weeks. At the time of completion of x-ray therapy, the serum bilirubin had risen to 12 mg. per cent on the one-minute direct determination and the total serum bilirubin was 16 mg. per cent. At the same time the cephalin flocculation was negative and the thymol turbidity was 4.2 units.

His condition continued to deteriorate and ascites became evident. A course of nitrogen mustard with ACTH was started. However the patient developed a fetor hepaticus and required codeine for increasing abdominal pain. He became very restless, getting in and out of bed frequently. At dawn of the eighty-fourth day after initial admission to the hospital he was noted to be having involuntary urinations, was confused and was found to be moaning restlessly and perspiring profusely. His skin became clammy and he finally expired at 8:15 a.m.

Discussion

Dr. Howard L. Horns: Since you have all read the protocol, perhaps we may consider the history in reverse order. This is the case of a middle-aged man who was deeply jaundiced when he died. The information available would indicate that the jaundice was not secondary to excessive hemolysis and therefore was a regurgitation jaundice. We need to decide whether the cause of regurgitation was intrahepatic or extrahepatic. He had dull, steady epigastric pain and at least one episode of sudden, severe pain in the right upper abdomen. The darkening of the urine at that time suggests some obstruction of the biliary tree. His past history contains two interesting items. He had a background of apparent hepatitis in 1921 and he had an alcoholic history which might be a basis for cirrhosis. The notation that his father died of leukemia is of interest in the light of the later histologic diagnosis of chronic lymphocytic leukemia in this man. The incidence of leukemia in other members of the families of persons having leukemia is a little higher than chance would allow. It is not clear whether this is an hereditary matter or not. An interesting suggestion has been made by Ludwig Gross[1] that this represents a lineal infection through the germ plasm.

The physical examination revealed a number of findings that are of some importance. The firm, non-tender nodes in the cervical region are of interest in view of the later histologic findings. I am not sure how to interpret the ptosis of the left lid. Certainly it was not a residual of his old facial palsy, since the one muscle which is spared in a Bell's palsy is the Levator palpebrae. Perhaps it was part of a Horner's Syndrome, secondary to the lymphadenopathy in the neck. I just don't know. The liver was described as being large, firm and tender. I would be interested in having a little more information about this tenderness. Diffuse tenderness might occur with chronic passive congestion, hepatitis, or metastatic malignancy. The protocol says the liver showed "possibly some nodularity below the edge on the right." That is a little difficult to interpret. There are two "weasel" words in there which make me wonder whether it was really nodular or not.

The laboratory findings do not help me a great deal. There are some that need comment. The high alkaline phosphatase in the absence of obstructive jaundice might suggest malignancy within the liver. There were two quite different values for the fecal urobilinogen that make one wonder whether there was some intermittent obstruction. The bromsulphalein retention was elevated. I won't argue with the histologic examination of the lymph nodes, although there is no evidence of chronic lymphocytic leukemia in the blood smear. This can occur. We have seen patients in whom the blood picture did not demonstrate chronic lymphocytic leukemia for a number of years after a lymph node biopsy had.

The patient had another episode of severe right upper quadrant abdominal pain while he was in the hospital, with a very strange blood picture at that time. In spite of fever, a leukopenia with a relative lymphocytosis was present. The subsequent right shoulder-strap pain would suggest that something was happening on the top of his liver, or at least adjacent to the diaphragm on that side. The spleen became palpable and he developed obvious icterus, with more than half the serum bilirubin present giving the direct reaction. Later, ascites developed and he expired in spite of treatment.

We have been given one diagnosis already and if we take that at face value we must ask if this whole picture can be explained on the basis of chronic lymphocytic leukemia. I suppose one would be foolish to say that it could not be, but it seems extremely unlikely on the basis of the clinical picture. The severe right upper quadrant pain is not characteristic of leukemic infiltration of the liver. The description of the tender liver is not particularly characteristic of a leukemic liver. The rather rapid development of jaundice is certainly not usual and the failure to respond to x-ray therapy is somewhat against a diagnosis of leukemia.

The question of parenchymal liver disease needs to be considered. He had two things in his background which might predispose to cirrhosis. One was the history of probable hepatitis in 1921 and the other was his alcoholism. However, he failed to show very much in the way of disordered liver function and there is little to support the diagnosis of ordinary cirrhosis here. Another thing that needs to be considered, in the presence of a big, tender liver, is the possibility of infiltration by a second malignancy, considering leukemia as the first malignancy. If such were present, a primary site would have to be considered. There are two primary malignancies within the abdomen that are very difficult to discover before metastases to the liver have occurred. One is carcinoma of the pancreas and I see nothing to suggest this diagnosis here. The second is carcinoma of the gall bladder. Carcinoma of the gall bladder spreads to the liver early and may produce extensive metastases there. Furthermore, at least 50 per cent of patients with carcinoma of the gall bladder are jaundiced at some time during the course of the disease. The possibility of an intermittent partial biliary obstruction would fit with this concept, either as a result of passing stones or perhaps because of the sloughing out of necrotic bits of tumor tissue. A further item that might support this diagnosis is the failure to demonstrate the gall bladder by x-ray. The terminal events fit with this concept. The ascites could be caused by extension of the tumor through the gall bladder wall, with seeding of the peritoneum.

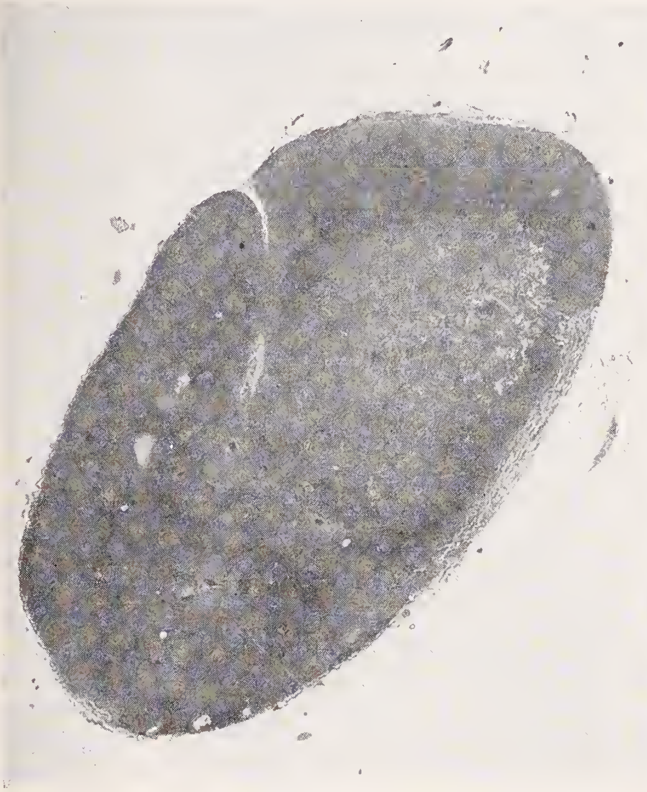

Fig. 1. Chronic lymphocytic leukemia in a lymph node.

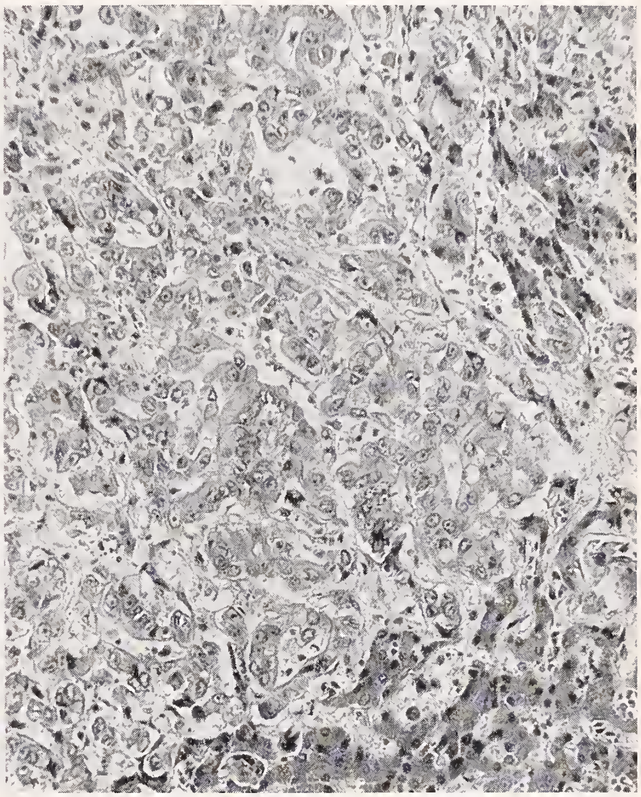

Fig. 2. Malignant cholangiocarcinoma of liver showing very anaplastic cells with pale, vesicular nuclei and a tendency to form glands. Normal hepatic parenchymal cells are at the bottom and to the right. Photography by Medical Illustration Service, VA Hospital, Minneapolis, Minnesota.

Dr. Horn's Diagnoses

1. Chronic lymphocytic leukemia, aleukemic phase.

2. Carcinoma of the gall bladder with extension to the liver.

Dr. E. B. Flink: Most of the physicians who participated in his care felt that the patient had carcinomatosis, either from the stomach or pancreas, and the lymph node biopsy was performed with this in mind. When the lymph node and the marrow revealed a lymphoma, all of the patient's findings were explained on this basis.

These are the diagnoses made by the medical students from today's protocol. One student put carcinoma of the ampulla as the primary diagnosis. Two other students thought that there was carcinoma in the region of the ampulla or in the pancreas itself. No one mentioned carcinoma of the gall bladder. Three students put leukemia as the only diagnosis. Two thought cirrhosis was the primary diagnosis. One suggested chronic pancreatitis.

Pathologic Findings

Dr. Gleason: There was severe jaundice. There were 5000 cc. of dark yellow ascitic fluid. There were 500 cc. of yellow fluid in the right pleural cavity but no pleural fluid was present on the left side. The heart and lungs appeared normal, except for a partial atelectasis of the right lower lobe. The liver weighed 3,700 grams. Approximately 80 per cent of this weight was due to tumor masses. These measured up to 5 cm. in diameter. The prosector felt that the intrahepatic tumor nodules were not randomly distributed but that they were much more numerous and somewhat larger in the right lobe of the liver, although they were scattered through all lobes. The gall bladder contained only 15 cc. of mucoid green bile. It obviously was not distended and it contained no tumor. No calculi were found in it or in the bile ducts. The common bile duct was not dilated, but was surrounded and compressed by nodes. We did not feel that the nodes were causing obstruction of the common bile duct. The spleen weighed 550 gms. and was somewhat firmer than normal, but the cut surface appeared normal. The pancreas weighed 150 grams. There were some round discrete masses of tumor in the region of the head of the pancreas but it was felt that they were metastases in lymph nodes and not tumor originating in the pancreas. There was a firm mass in the right adrenal which was thought to be tumor also. The alimentary tract was entirely normal from the esophagus to the rectum, but the feces were white. There was a moderate generalized enlargement of the lymph nodes. None of these were over 2 cm. in diameter. Most of them were about 1 cm. in diameter and were relatively soft and just somewhat more prominent than usual. The remainder of the gross examination was normal.

On microscopic examination, it can be seen that the lymph nodes contain almost solid lymphoid tissue, most of the normal architecture being obliterated (Fig. 1). The patient did have chronic lymphatic leukemia. On looking at the slide from the spleen, it is more difficult to be sure, unless one knows that the spleen was large and unless one knows the age of the patient. A man of this age would not ordinarily have much lymphoid tissue in his spleen and it should not be so large. If we were shown this spleen with no additional information, I am afraid we would probably call it normal. The bone marrow from the autopsy is not much more convincing for lymphocytic leukemia than it was during life, but the lymph nodes are sufficient for the diagnosis. The lymph nodes from the porta hepatis were also leukemic, but in addition contained metastatic tumor which was a poorly differentiated but easily recognizable adenocarcinoma. Sections of the common bile duct reveal a normal mucosa but carcinoma cells can be identified within its perimuscular lymphatics. We are interpreting this finding as evidence for metastases to the outer portion of the wall of the common bile duct, via lymphatics from the liver. Sections from the liver reveal no cirrhosis and no appreciable leukemic infiltrate. Many sections, however, show the same poorly differentiated adenocarcinoma (Fig. 2). The right adrenal also contained some metastatic adenocarcinoma. We were also surprised to find small nests of tumor cells in the sections from the lungs. In attempting to piece the entire picture together (considering the absence of a demonstrable primary site in the pancreatic-biliary ductal system or in the gastrointestinal tract) we have come to the conclusion that this case is actually that of a primary carcinoma of the liver, of the cholangiocarcinoma type. This is a man who did have chronic lymphocytic leukemia, just as his father did, but who died because of a malignant cholangiocarcinoma of the liver.

Dr. E. T. Bell: I might say that primary carcinoma of the liver isn't as rare as we used to think. Approximately 1.5 per cent to 2 per cent of all fatal malignancies in our autopsies have been primary in the liver. The two major types of primary hepatic malignancies are the rare cholangiocarcinomas, of which today's case is an example, and the hepatomas. The cholangiocarcinomas are believed to be derived from the small intrahepatic biliary ducts and have no particular relation to cirrhosis. The malignant hepatomas resemble the hepatic parenchymal cells and are more common in cirrhosis.[2]

Dr. Flink: Dr. Horns came about as close as he could have come, and I think he should be congratulated for his straightforward thinking.

Dr. Bell: He should be congratulated for recognizing that there were two diseases and that the leukemia was not the cause of death. I think that was quite an achievement.

Dr. Flink: Would you care to make any additional comments, Dr. Horns?

Dr. Horns: Carcinoma of the ampulla was suggested by some of the medical students. This entity presents an over-all picture that sometimes allows one to diagnose its presence before surgery. It is a lesion that does produce painless jaundice, whereas carcinomas of the pancreas usually do not. The jaundice is often intermittent because of the periodic sloughing of tumor tissue. Blood may be found in the stool or in aspirates of the duodenal contents, for the same reason. Therefore because of the recurrent right upper quadrant pain and because of the absence of blood in the stool, I did not feel that this was the most likely diagnosis.

Dr. Gleason: I would like to say a few words about the frequency of common bile duct obstruction caused by metastatic tumor in lymph nodes. This mechanism is not as common a cause of jaundice as many physicians believe. A review of our cross-files on over 1100 autopsies discloses that there were seventy-eight cases of jaundice associated with malignancy of some kind. Only eight of these cases were due to obstruction of the common bile duct by malignancies, excluding malignancy of the pancreas or of the bile duct itself. Three of the eight were due to carcinoma of the stomach. Gastric tumors frequently grow into the porta and will attack the duct quickly. There was one carcinoma of the testis but this wasn't a case of compression of the duct by nodes but an actual metastasis into the wall of the common bile duct, with subsequent obstruction. There was one carcinoma of the colon which had metastasized to the nodes around the common bile duct, compressing it and causing jaundice. There was one bronchogenic carcinoma, one tumor of the bladder and one case of Hodgkin's disease. If we exclude the gastric cancers, as I would like to, we find that only five out of seventy-eight cases of jaundice associated with malignancy were due to obstruction of the common bile duct by metastatic tumor. The majority of the remaining

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Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

THE ROLE OF THE BASIC SCIENCES IN MEDICINE

Medicine and Applied Science

It was in 1892 at the University of Minnesota that the then Professor of Medicine in the new Johns Hopkins University, later Sir William Osler, presented his well-known address on "Student and Teacher." In it, he outlined his quartet of virtues of the good physician: "The Art of Detachment, the Virtue of Method, the Quality of Thoroughness, and the Grace of Humility." In discussing the Virtue of Method he complained that, "In the practice of medicine, where our strength should be lies our great weakness." He referred to the fact that at the time of his address "the practice of medicine is an art, based on science," but that in matters of practical importance to the physician there was little or no exact science to be applied.

Osler apparently expected great changes to occur because he commented that "when fifty or sixty years hence some historian traces the development of the profession in this country, he will dwell on the notable achievements, on the great discoveries—," but it is likely that he never dreamed the practice of medicine would be changed as radically as it has been in the sixty-five years since he spoke. At that time, the great advances in medical science over medieval medicine were largely limited to the fields of morbid anatomy and descriptive bacteriology. Such scholars as Rokitanski, Virchow, Pasteur and Koch had laid the foundations for those fields. But Paul Ehrlich had just begun to work, and the very real advances in physiology and physiological chemistry made by Claude Bernard, Johannes Müller, Wöhler and others had made little impact upon medical practice because their studies did not offer great practical applications.

Actually, Claude Bernard saw very clearly more than a hundred years ago the inevitability of the developments that have occurred in the past century. His lucid philosophical essays on "Experimental Medicine" presented the pattern which has been followed to bring medicine out of the realm of guesswork and into the area of applied science. His outlines of the scientific method could not be improved upon appreciably today, except in the utilization of newer tools of mathematics, physics and chemistry. But in the last decade of the nineteenth century, the development of the scientific basis of medical practice had only begun. The developments of the last half century in scientific medicine have been significant, but it would be a great mistake to think, either that they have made medicine into an exact science, or that progress in medical knowledge has reached a plateau. Actually, the areas of our ignorance about many aspects of physiology, biochemistry, microbiology, pharmacodynamics, and even of morphology, in health and in disease are greater than the areas of our knowledge. The enormity of our ignorance has become clearer to us as we have pushed the frontiers of knowledge forward, because as we probe into the unexplored territory we see how vast it is. And as we see the accelerating pace of research into this vast unknown, we cannot fail to realize that the future is bound to see even more remarkable progress toward the control of disease and degenerative processes than we have witnessed in the last half century.

The physician in the future will inevitably be more and more an applied scientist because progressively more of his diagnosis and his therapy, both surgical and medical, will involve the use of quantitative measurements whose interpretations depend upon knowledge of the sciences basic to medicine. Fifty years ago, the physician who used a microscope was a curiosity. Today, a physician who does not utilize microscopy, chemistry, radiology and physiology in diagnosis and in control of therapy would be guilty of malpractice.

To be quite personal, when I began the study of medicine thirty-five years ago, the only very "practical" preclinical sciences, according to many of our clinical teachers, were gross anatomy and pathology. The situation is changed today, not because morphology is less important in any absolute sense, but simply because the other basic sciences have likewise become indispensible to enlightened medical practice. This situation has come about because clinical investigation and basic science investigation of the disease and degenerative processes *in life* have given the clinician powerful new tools which most physicians did not dream of having as little as thirty-five years ago.

Maurice B. Visscher, M.D.
Department of Physiology
University of Minnesota

THE CLINICAL PSYCHOLOGIST

On the national scene, the clinical psychologist, as of 1957, is a well-accepted member of the faculties of the medical schools, and he will ordinarily be found in the Department of Psychiatry. From the standpoint of academic training, he will either hold a Ph.D. degree or be in the process of obtaining it. The Ph.D program requires a minimum of eight years of college and hence is as long a training program as that of the medical student. The Ph.D. curriculum is occupied with basic aspects of learning theory, statistics, research methodology, and clinical experience. It contains some of the basic courses which medical students take, such as neuroanatomy. The candidate must conduct a piece of original research which meets the requirements of the graduate school, and this is published as the Ph.D. thesis. One year of the curriculum is spent under medical supervision in the hospital and out-patient clinic.

This training program tends to produce a man whose major interest is in research or academic life, and approximately two-thirds of the Ph.D. holders from Minnesota are found as full-time members of university faculties from New York to California. Many are occupied in full-time research in mental health, such as investigators into human conditioning, psychopharmacology, and testing aimed at evaluating various therapies. Others devote major time to teaching and the clinical work of testing patients in hospital and clinic. The clinical psychologist has become an invaluable member of the medical school faculty.

As the clinical psychologist has come to deal with sick people and has attempted to apply his knowledge for their benefit, it is understandable that he has developed interest in treatment. Some clinical psychologists have developed their major interest in psychotherapy and have demonstrated excellent aptitude for it. Although not the pattern in Minnesota, other states, New York in particular, have seen the clinical psychologist enter private practice and undertake the therapy of neuroses and personality disorders.

It is in this particular area, the solo practice of psychotherapy without medical supervision, that the clinical psychologist has found difficulties with the medical profession. It would be unrealistic to state otherwise, and this is an issue still largely unsettled. Medicine, with the American Psychiatric Association as its spokesman, has taken the stand that the treatment of the sick is a medical responsibility and that while certain aspects of therapy may be delegated to non-medical personnel under supervision, the primary responsibility rests with the physician. To treat sick people without a medical license constitutes an illegal act.

The clinical psychologist on his side will argue that this is true but that personality disorders do not constitute illness as intended by the statutes pertaining to medical practice. Were this so, it would also be illegal for the clergyman, the lawyer, and the helpful friend to give counsel to an emotionally disturbed person. Further, the clinical psychologist is better prepared often to practice psychotherapy than is the average doctor of medicine, unless he too has taken specialized training. Both groups are in full agreement that the conflict is not related to economics but to principle, for there are not nearly enough physicians or anyone else specially trained to begin to give help to the mass of people with personality and emotional disorders.

From whichever side of the fence one may view this personally, there is no question that a conflict about the principles involved exists. The status quo cannot remain, but the pattern that will evolve remains for the future to decide. The American Psychiatric Association and the American Psychological Association have a joint committee working actively on the problem. Some guesses as to future developments will be the subject of the succeeding editorial.

Donald W. Hastings, M.D.
University of Minnesota

A CLINICIAN READS THE JOURNALS ON FATS AND ATHEROSCLEROSIS

The reports in the *Journal of the American Medical Association* of August 24, 1957, of the symposium held in New Orleans, bring the discussion up to date. From the reports regarding atherogenesis as it relates to fat metabolism newer points of view emerge. The function of cholesterol is still an unsolved problem. It has been accused and maligned as a culprit without foundation. The agitation, however, has stimulated the biochemist to more intensive study of lipid metabolism. This has led to the idea that the whole subject of nutrition must be re-evaluated. Not until all the lipids have been correlated in the metabolism of fat—and its functions in the body; not until fats in relation to protein and carbohydrates metabolism have been restudied will the problem of atherogenesis be clarified. That atheroma is a part and result of nutrition is obvious. So is immunity a part of nutrition. The aging process is also one of nutrition.

Comparing statistically the atherosclerosis of the coronary arteries in the different countries with that of the United States of America and attributing the difference to the amount of fat and the quality of the fat ingested is ingenious. To explain the difference to be based just on the fat supply in the respective food in the various countries studied is not quite enough of an explanation to satisfy a goodly number of physicians. This method of study is an approach reminding one of the old problem of blood pressure variations in Asiatics when compared with western populations.

Letters to the Editor in the *Lancet* of May 12 and again June 23, 1956, not only antidate but anticipate the discussions in New Orleans.

In all these talks, there has been no mention of the lungs as a factor in fat metabolism. Let me call your attention to a fundamental anatomic fact. The thoracic duct is found in all mammals. There must be some definite reason why the chyle (fat) from the intestines is shunted to the lungs before it reaches the general circulation. Furthermore, the lipid levels in the blood are repumped through the lungs at each cardiac cycle. What does this do to the fat moieties in the plasma? Does this pulsatile activity in the lungs add or subtract something from these fat levels?

When the thoracic duct is ligated or obstructed by trauma, the lack of continuity of the duct does not appear to have any influence on the health of the individual. However, it must be recalled that immediately after an obstruction of the duct numerous collaterals are established. This effort to establish collaterals so quickly, to my mind emphasizes the importance of the delivery of the chyle to the lungs. Then the steroids are chemically a branch of the fat family. I am alluding to sex hormones and the adrenal cortex group. Have they any play in the level of the different fats circulating in the blood? Have they any influence in maintaining the health of the arteries; or in hastening their degenerative changes? To restate the thought, are they to be considered in fat metabolism and its physiology?

These features of fat physiology must be studied and added to the fast accumulation of observations on fat in the circulation and in the tissues.

To come back to cholesterol: It is my opinion cholesterol has a reparative function rather than that of a pathogenic factor.

Henry L. Ulrich, M.D.

FOR REFERENCE SAKE

Much of all the work done in a medical library is ultimately for reference sake. Books are selected, ordered and processed with this service in mind. What questions will they answer, or what gap in the collection will they cover, are considerations that are foremost in the mind of the librarian as she weighs needs and usage against the rising cost of books and journals.

A day, reference-wise, may develop something like this. The telephone rings. An unidentified caller wants to know the name and address of the psychiatric hospital in Smith, Texas. This is a simple question that can be answered in a few seconds with the help of the current *Hospital Guide*. Reference question No. 1 is recorded, the kick-off to a reference-centered day.

As the librarian starts checking the daily journal mail, a routine process goes into operation. Doctor Reilly will want to be alerted to the article on atmospheric pressures and their relation to oxygenation in the *Journal of Clinical Investigation*. The librarian makes out a form contact slip, putting name, journal, title and pages on it, places it in an envelope and addresses it to the doctor's office. And here is an article in *Surgery* on vinyl graphs. This will interest the resident in research who is currently doing work in this field. Thus, journal after journal is scanned and articles evaluated as to potential uses. By anticipating the needs of her clientele, the librarian is not only saving time for the hurried practitioner or student, but she is saving the time of the library staff. And she is speeding up the progress that comes from close association with recent medical thinking as it appears in the pages of current publications.

Her attention is eventually transferred from the stack of journals before her to an awareness that someone is looking for help. An over-busy intern must discuss a case at the Thursday morning medical conference. He needs material on fluid dynamics and congestive heart failure. He knows what references he wants and she selects them for him from the stacks, all but two. These will have to be borrowed from the near-by medical society library as soon as a "runner" is available.

Back at the periodicals another phase of the service, again a preparatory or anticipatory step to reference work, is begun. More than half of the journals received in the library are indexed. Each article considered of importance to the library's clientele is entered on a 3 x 5 inch slip and assigned a subject heading—one that patrons using the index may look for readily. All slips are then arranged in alphabetic order and filed according to subject. This is the quickest approach to the most recent material in the library's collection. The time lag of over two years removes the *Quarterly Cumulative Index Medicus* from the category of a current index. Entries in the *Current List of Medical Literature* are necessarily at least three months behind the current issue of a journal, and the index is awkward to use. Many librarians resort to their own indexing as an expediency. In some urban communities, the indexing becomes a joint project among several medical libraries in the area. Most librarians know that the time expended in bringing recent material to the attention of the medical profession is more than compensated for when the actual search for recent articles is on. Every one likes his information with the ink still wet on it.

The request for material on a special subject, covering a range of five to ten years, is probably the most common of all in a medium to large size library, especially in hospital libraries with an active teaching staff and program. A paper or a lecture is reason for a review of the literature which the conscientious physician seldom overlooks. The least frequent request in a non-university library, research libraries also excepted, is the one for a complete review of all literature in a selected field. If a smaller library undertakes this task, it must lean heavily on interlibrary loans, not only from other medical libraries within the community, but from libraries at greater distances. Usually, the patron in need of extensive references applies to the largest library in his locality, knowing that both the index and reference holdings will be more comprehensive.

The traditional definition of a librarian as one who does not know all the answers but knows where the answers are found is based on the reference concept of library service. Again, taking into account human factors—electronic knowledge searchers have not yet arrived—it does happen that reference service does not always jell. The medical librarian who humorously related the trials of being the "handmaid of the medical profession" in a poem published in *Lancet,* March, 1950, directs her complaint to the problem of the indefinite request which, especially without the aid of indexes, can be both time-consuming and frustrating. Expressing her patron's vagueness as to the title of the article wanted, the time it was written, the journal in which it was published and its authorship, she writes:

> An infection found in beavers was transmitted by retrievers,
> And carelessly contracted by a vet,
> While the organism, injected in a toad in Timbuktu
> Was recovered from a tadpole in Tibet.
> Now was it in the LANCET in 1892?
> Or the B.M.J. in 1923?
> Or maybe PATH. AND BACT.? No, I can't be more exact,
> And I think the name was Smit, or Smut, or Smee?

Fortunately, it does not happen often that request details are not spelled out. When it does happen, and it does in the best regulated setups, a smooth-flowing reference service is temporarily side-tracked. Sometimes the "Smits" and the "Smees" lead into hours of blind alleys with the staff in pursuit, sleeves rolled up, and determined not to be beaten by an elusive reference. These excursions are a challenge and bring variety to a day's work. Memory frailties are not limited to one side of the desk only; a reference day tapers off with an even balance of both. No man-controlled system could be otherwise.

Sister Teresa Louise
Medical Librarian

YOUR WILL

Medicine has made great strides in our time, but up to now no therapy has been perfected which will assure the continuance of our poor bodies in perpetuity—and this applies to skilled and erudite physicians and surgeons as well as to just ordinary mortals. In time death will come, and attending it, if there is an estate, will be a bundle of problems for the family, including administration, the payment of funeral expenses, debts and claims, the division of the property, and the ever-present handmaiden, taxes.

Usually, an estate consists of all assets less liabilities as determined at the date of death. Some lucky few inherit estates, others "strike it rich," but most people acquire one by building it. Do you have an estate? Yes, certainly, and if you will take a few short minutes to tally up the score, you will be surprised at its size. For instance, in determining federal estate taxes, the following items are examples of what must be considered as part of your estate:

- Life insurance on your life (unless owned by one other than yourself)
- Joint tenancy properties (here we bunch homestead, jointly held securities, U. S. savings bonds payable "and/or" or "payable on death to............................," and joint bank accounts, including savings accounts, except to the extent contributions by the surviving joint tenant can be established)
- Real estate owned individually by you
- Securities owned individually by you
- Your personal cash, including bank deposits
- Professional equipment, office equipment and accounts receivable of your practice
- Household goods, wearing apparel, hunting and fishing equipment, jewelry, automobiles, and other tangible items

Everything above $60,000.00 in value, after deducting administration expenses, debts, funeral bills and what passes to your wife, if you have one (but not exceeding one-half of the gross estate) is subject to the federal estate tax, and the rates mount pretty rapidly.

If you have anything, you have an estate. This estate deserves careful consideration at your hands, and at the hands of expert advisers. Here preventive measures can determine distribution in such a fashion that your family will receive protection, and by planning within the tax structures, you can preserve your estate at a higher dollar value.

Probably the primary question in your mind now is, "Do I need a will?" What you leave upon death must be distributed somehow. You should take advantage of the various means provided—such as a will, or joint tenancy deeds, or insurance agreements, or trust instruments, or a combination of these means depending on the particular needs and problems which may confront you. If you die intestate, meaning without a will, the law decides how your property will be divided and how it will descend. Sometimes this "law will" accomplishes the desired end, but most times it does not. Like a size 48 suit, it may fit someone, but it doesn't fit everyone.

In Minnesota, for instance, except for a few minor variations, this is what will happen to your property if you die intestate. The home will descend to your children, with the right in your wife to occupy it and use it as long as she lives. The rest of your property will pass as follows:

- All to the wife, if there are no children
- One-half to the wife and one-half to the child, if there is one child surviving
- One-third to the wife and two-thirds to the children if more than one child survives
- If no wife survives, all to the children with the issue of a deceased child taking the share which the parent would have taken (by right of representation)
- If there is no wife or issue, to your father and mother in equal shares, and if only one survives, to the survivor
- If there is no wife, surviving issue, father or mother, then to your brothers and sisters, and to the issue of deceased brothers and sisters the share which the deceased brother or sister would have taken
- If none of the foregoing survives, to the next of kin, and if there is no wife and no kindred, all of the property will pass to the State of Minnesota.

Whether or not the jacket fits can best be determined by your legal adviser after you have given him all the facts respecting your assets, your family and other objects of your bounty, and your hopes and aspirations with respect to them. Individual situations vary, and your particular problems very likely will differ materially from those of others. Do not trust to chance, but consult a good lawyer and find out what you should do about planning an estate.

A Minnesota Lawyer

SPACE RADIATION HAZARDS

The band of extremely intense radiation surrounding the earth at a distance of 600 to some thousands of miles does not pose a threat of quick death from radiation to men traveling in space, according to Dr. Harvey M. Patt of the Argonne National Laboratory, Lemont, Ill. This judgment was based on animal studies and reports of geiger counter measurements made by U. S. satellites. The accumulation of radiation by a body in this band will, in a few hours, approximate the established weekly tolerance level. This does not, however, constitute a threat of acute radiation poisoning. If repeated often or for an extended period, the possibility of pernicious effects does exist—both for the individual exposed and his progeny.—*New York Academy of Sciences,* May 2, 1958.

President's Letter

HOSPITAL BEDS

Do you have trouble getting a bed for your patient in your hospital? Where is the physician who does not? We all know that hospitals are overcrowded, and that there are many reasons why this is so. During the depression in the 'thirties, hospitals were partly empty. Prepayment plans for hospital expenses began then, primarily Blue Cross. Then came World War II and prosperity, and hospitals which were not essential to the war effort were unable to expand. After the war, the enormous pent-up demand for building made hospital construction difficult. Inflation increased; building costs rose. Overcrowding became almost unbearable, however, and expansion became imperative. The Federal Government recognized the need, and the Hill-Burton law was passed. This was designed primarily to help the rural areas where hospitals were few and far between. As a result, many hospitals have been built in the smaller cities and towns. The Ford Foundation, three years ago, distributed $200 million to the hospitals of the country. While this is an enormous amount to come from a private foundation, especially with no strings attached (except that it be used for construction and not for maintenance), still in comparison to the need, it was a drop in the bucket, and was primarily a stimulus to get on with the job.

In Minnesota, hospitals have been built all over the state, but the need for hospital beds still far surpasses the present supply. The figures of daily occupancy of hospital beds have become almost meaningless. Reports of occupancy are regularly 100 to 120 per cent. This means that beds are placed where they do not belong; two beds in single rooms, three beds in two-bed rooms, in corridors, and in rooms which should serve other purposes. This also means that laboratories are overcrowded and therefore inefficient and unable to add necessary new procedures to keep up with advances in scientific medicine. Operating rooms are working overtime. X-ray departments are squeezed, and patients wait in the halls. Just as important, kitchens built to serve 100 patients are serving 120, and adding extra meals for the staff. Laundries are putting out more than their intended load. All this is not just inconvenient. Surely the crowding, the hurry and the confusion add to the problems of infection, just now, staphylococcus infection.

Of all the people in the state, physicians are closest to this situation. We spend one-third to one-half of our working hours in hospitals. We know the difficulties. We work to help solve them, and we contribute time, effort and thought to their aid. Unreasonable as it is, there is still gossip around the state that physicians are not doing enough. We have the reputation of being rich, unjustified it is true, but the reputation is there. As a whole, we really are well to do. It would be well to do what we can—and a little more—financially and as salesmen. The United Hospital Fund in Minneapolis needs more contributions from physicians, and could use a lot of sales effort by ourselves among patients and friends. There are drives for funds in Saint Paul and Duluth which need help. The Minnesota State Board of Health states that over the past ten years an average of $19.3 million has been spent on hospitals each year. Their conservative estimate of needed expenditures for each of the next ten years to liquidate existing bed deficits, provide for increasing population, and replace and remodel obsolete facilities is $36 million. This is for each year, not the total for ten years. At our present rate, we are falling farther behind, not catching up. Funds are needed from every possible source, not only federal and state, but county and municipal, corporation and foundation and, perhaps most important, personal and individual. If we have given the limit ourselves, let's convince our well-to-do friends and patients of the need, so that they may have hospital beds when they need them, and we can give them the kind of care they should have, the kind we can give only in the proper place.

Horatio B Sweetser

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

AMA LEGAL HEAD COMMENTS ON MEDICAL LIABILITY ACTIONS

Professional liability has been the subject of a two-year study by the Law Department of the American Medical Association. In charge of the study was C. Joseph Stetler, Director of the American Medical Association Law Department. The study was based on a 5 per cent random sampling of AMA membership. Of this group, 80 per cent responded. These are the findings:

Of all AMA physician members in the United States, 14 per cent have had a medical professional liability claim or suit brought against them. California is highest with one out of four, and South Carolina lowest with less than one out of every thirty-three.

Of all living United States physicians, approximately 18,500 have had a claim or suit brought against them at some time. But 40 per cent of these claims and suits have either been dropped or decided in favor of the physician. Of physicians who have faced claims, 87 per cent have had one; 10 per cent had two; 2 per cent had three; and 1 per cent had four.

Of the doctors questioned, 29 per cent thought incidence of such claims had increased over the past five years. No change was noted by 40 per cent.

The full-time specialist is most frequently the defendant. This is not surprising because the average claimant probably assumes that the specialist has a higher income and carries more insurance.

Although only 22 per cent of the physician population was board-certified in 1956, 29 per cent of those claimed against or sued were certified at the time the allegedly negligent act was performed. The reason for the slightly higher percentage probably lies in the relative complexity of the medical problems they handle.

The recent medical school graduate is by no means the most frequently sued doctor. The median length of practice in the sued group is 13.4 years.

The survey explodes some myths regarding liability insurance. We have heard for some time how difficult it is to obtain coverage, but more than nine out of ten doctors indicated they have had no trouble. Even those who have already had a claim or suit do not have difficulty. Of doctors who have faced claims, 93 per cent were carrying insurance.

Is the cost of this insurance excessive? Prior to the study AMA officials would have said most physicians believe it is; the study was prompted in part by such complaints. But 56 per cent thought their coverage cost was reasonable.

The primary reason for claims and suits was a poor operative result in 24 per cent of the cases reviewed; poor medical results in 20 per cent; diagnostic errors in 10 per cent; and foreign bodies left in the patient at operation in 9 per cent. Other categories represented 8 per cent or less of the cases.

As for secondary reasons, 27 per cent are under the direct control of the profession and include careless comment about another doctor and misdiagnosis or poor results in situations in which the patient had another disease or disability. Another 22 per cent include instances of pressure or influence by outsiders such as an attorney or relative of the patient. The patient himself is responsible for 25 per cent of the secondary causes.

A study of the fields of medicine involved shows that 31 per cent of the allegedly negligent acts occurred in surgery, 20 per cent in medicine, 20 per cent in orthopedics, 12 per cent in obstetrics and gynecology and 17 per cent in other fields.

Where did the allegedly negligent act occur? In the hospital in 67 per cent of the cases, in the physician's office in 24 per cent, in the patient's home in 6 per cent and at work or elsewhere in 3 per cent.

The median age of the claimant is 34.4 years; female claimants predominate.

One of the most important questions was whether the claimants actually received a money award. In 45 per cent of the claims and suits, the claimants obtained money from the physician, and in 40 per cent they did not. In the claims stage (prior to suit), the number receiving some money was nearly twice that of those who did not. In the suit stage, the claimants who failed to collect outnumbered those who did, two to one.

Effective prevention programs must be started at all levels, aimed at conducting a practice which will minimize professional errors and reduce the likelihood of having claims filed.

MINNEAPOLIS FIRM ANSWERS FEDERAL TRADE COMMISSION CHARGES

The Qualitone Hearing Aid Company, Inc., a Minneapolis contact lens and hearing aid firm, has filed a reply with the Federal Trade Commission defending its advertising claims for "Hidden Ear" and "Stereophonic Optical Ear."

Recently the FTC also filed a similar charge against Klear Vision Contact Lens Specialists, Inc., of New York City. The latter firm has also denied FTC charges.

The commission also received a statement of denial from Mytinger & Casselberry, Inc., of Long Beach, California, whose business practices in marketing vitamin and mineral supplements have been branded monopolistic and illegal by the Federal Trade Commission.

DRUG SAMPLE SURVEY UNDER WAY

A study to determine what happens to drug samples sent to physicians has been announced. The survey is being conducted by Taylor, Hawkins and Lee, Inc., a medical marketing research firm, in co-operation with the pharmaceutical industry and the AMA advertising department. The program is designed to provide basic facts on the uses to which drug samples are being put by practicing physicians.

Results of the study being conducted on a nationwide basis during the spring and summer will be available by next fall to provide practical guidance in the use of samples as a market technique.

Many physicians contribute their samples to such organizations as World Medical Relief which provides drugs, instruments, medical equipment and magazines to various mission fields.

MEDICINE NEEDS LEADERSHIP—BERRY

Organized medicine ought to shed its role as the constant dissenter, the inevitable *nyet* when proposals are advanced which embody departures from ordinary practice in payment for medical services. This, in substance, was the nature of remarks made recently by Assistant Secretary of Defense, Frank B. Berry, while delivering the J. Shelton Horsley Memorial Lecture at the Medical College of Virginia. Dr. Berry further points out that leadership by medicine is needed, not unquestioning protection of the status quo.

HIGH MEDICAL COSTS UNDER FIRE

Congress is set to take action to curb the spiralling costs of medical care.

Already investigation is under way to ascertain methods used by the Bureau of Labor in compiling statistics to determine the costs of medical care published in the bureau's monthly and quarterly price indexes.

Price indexes published by the bureau are factors used to determine the raising or lowering of wages. Similarly, index findings dealing with medical care are guides used by Health Insurance companies and Federal agencies to negotiate medical care and hospitalization contracts between Federal agencies and private carriers.

In 1957, the government's average index for "medical care" was 138.0. This means that health services, hospitalization and certain drugs, pooled under one heading, were 38 per cent higher than they were in the 1947-49 base period. For all consumer items put together (food, clothing, housing, medical care), the rise was 20.2 per cent. Thus, if the "medical care" methodology and computations are accurate, the rate of increase here is *nearly two times that of the over-all list.*

FREE CHOICE OF MEDICARE PHYSICIANS THREATENED

Medicare, as it presently exists, will receive a thorough going over before Congress lets go of the purse strings on an $80 million request for funds for the next fiscal year. It now appears that the Defense Department may exercise its prerogative to restrict military dependents' privilege of choice of private doctors and hospitals. Congress is receiving support from several branches of the Armed Forces in its "get tough" approach to medicare.

Navy Spokesman Comments

Rear Admiral F. P. Gilmore, Assistant Chief of Navy's BuMed, in closed session of the House Appropriations Committee, commenting on the cost of the program, admitted to that group that the Medicare program has failed to live up to promise.

He presented these figures: In the fiscal year 1958-59, the Navy will have responsibility for

692,000 dependents of active duty personnel. The budgetary earmarking for medical care in non-naval facilities — mainly for dependents — is $33,031,000, or 29 per cent of the Navy's medical care budget. Each dependent in a civilian hospital is costing the government an average of $50.25 a day. This includes the physician's bills but excludes the patient's share of expenses.

It was further pointed out that in 1957, the first full-year in which Medicare was in effect, admissions to naval hospitals declined 21 per cent, compared with the previous year (107,200 to 84,300).

Air Corps Comments

Major General Olin F. McIllnay, Deputy Surgeon General of the U. S. Air Force, in testimony before the House Appropriations Committee, predicted that the Air Corps will take "definite action" to abridge dependents' free choice between military and civilian (fee basis) medical and hospital care.

He stated:

> "We believe that when Congress authorizes us to expend funds and construct hospitals, and when we have to staff those hospitals to provide for care for the military anyway, and the facility is there, that in those circumstances the provision of dependent care which could be provided for in the military facility but is received in a civilian facility results in an additional expense to the government. In other words, the government is in a sense paying twice for the same thing."

According to Major General McIllnay, civilian care of dependents is not only more expensive, but it is having an inimical effect on professional training programs in Air Force hospitals. USAF has the biggest Medicare budget for the fiscal year beginning July 1, calling for $28,220.00 for compensation of physicians and hospitals. This compares with Navy's $23,494,400; Army's $18,532,000, and $1,660.00 for U. S. Public Health Service.

AMA General Manager Comments

In the recent letter to the Honorable George H. Mahon, chairman of the Subcommittee on Department of Defense Appropriations, House of Representatives, Washington, D. C., Dr. J. L. Blasingame, General Manager, American Medical Association, pointed out reasons why the existing system of free choice of Medicare physicians should not be abandoned. He commented:

> "The American Medical Association does not agree that tax money would necessarily be saved if dependents of service personnel are denied a free choice. Furthermore, we are convinced that elimination of free choice would result in lessening the amount of authorized medical care now available to and received by the dependents.
>
> "It is difficult for many reasons to make an accurate cost comparison between civilian and military medical care and hospitalization. Depreciation costs, fringe benefits, as well as the expenses involved in recruitment, training, etc., are not included in computing the cost of military medical care. In this connection, we would respectfully invite your attention to the statement of Dr. Hugh Hussey before the Senate Subcommittee on Defense Appropriations on June 19, 1957. In addition, any attempt at a comparison of costs must also take into consideration the types of cases involved. The Medicare program under Title II of P.L. 569, 84th Congress, has dealt predominately with obstetrical cases which have a relatively high cost per hospital day.
>
> "Many of the costs to date in the administration of P.L. 569 have been of a non-recurring nature and were necessary in contracting for civilian care through the medical associations, hospitals and their designated fiscal agencies. A significant part of this cost was for informing all parties and agencies concerned. Any change at this time would only entail further disruption in administrative mechanisms and would result in the total loss of the non-recurring expenses of inaugurating the program.
>
> "To eliminate the free choice provision in the law, as spelled out in the 'implementing directives,' would necessitate in many instances the exclusive utilization of military and naval facilities staffed by physicians in the uniformed services. Should this occur, and should the armed services take over the civilian phase of Medicare, or a substantial part of it, an increased demand for physicians in uniform would also undoubtedly result—a demand which could probably be met only by reactivation of discriminatory draft legislation."

Dr. Blasingame further pointed out that it should be emphasized that dependents of those in the uniformed services are civilians and should seek their medical treatment through civilian channels if we are to preserve the beneficial aspects of our present system of providing medical care and to guard against further maldistribution of professional health personnel.

Texas Vetoes Medicare

Texas physicians, at their annual meeting recently, voted by a margin of better than two to one instructing the officers of the Texas Medical Association *not* to enter into any contract with a governmental agency. This means that after May 1, 1958, there exists no Medicare contract between the Texas Medical Association and the government. After careful appraisal and lengthy and spirited discussion, it was agreed by an overwhelming majority that renewal of the Medicare contract would continue to give subtle approval to the principle of government controlled medicine and government setting of fees. The Texas delegates voted unanimously to request its AMA delegates to initiate steps designed to bring about either the repeal of Public Law 569 (Medicare) or its modification so as to "provide medical care for the dependents of military personnel through indemnity programs underwritten by voluntary

(Continued on Page 418)

The World Health Organization

A Decade of Achievement in International Health Programs

The year 1958 marks the end of the first decade of the World Health Organization, the first truly global international health agency. Mankind has long recognized that disease knows no boundary. We are now learning that organization for health is also without barriers.

Delegates reviewed the accomplishments of the eighty-eight member nations of the WHO in special anniversary commemorative sessions as part of the eleventh annual World Health Assembly held in Minneapolis, May 26 to June 14. The record is an impressive one, even though much remains to be done.

During the last ten years, intensified national and international efforts have forced communicable diseases into retreat in many areas of the world. In 1948, it was estimated that malaria caused three million deaths each year out of 300 million cases. Today, the number of deaths has been reduced to 1.5 millions and the number of cases to 150 millions. This reduction has been due to mass campaigns assisted and encouraged by WHO in which residual insecticides like DDT and dieldrin were used to cut the man-mosquito-man cycle of infection. However, from 1951 onward, malaria-carrying mosquitoes began to show signs of resistance to insecticides. The policy of replacing routine malaria control with malaria eradication was instituted in 1955 and, by the end of 1957, malaria eradication had been achieved in nine countries or territories and is under way in fifty-one others.

In addition to the campaign against malaria, WHO wages war on forty other communicable diseases.

Methods of approach, similar to those used in the fight against malaria, are now being employed against smallpox. It is now possible to eradicate smallpox from a country by using a new stable vaccine produced as a consequence of studies promoted by WHO. Continuing work on the most stable vaccine showed that after two years' exposure at 45° C, it still produced 100 per cent successful vaccinations in man. Eradication programs have already started in some countries, particularly in Latin America.

Interesting new developments in regard to the treatment of trachoma have also resulted from WHO-assisted studies in Taiwan and Morocco. Pilot projects in these areas indicate that this serious eye disease, which afflicts 15 per cent of the world's population, may eventually be controlled by means of mass treatment with antibiotics. Through the use of aureomycin and terramycin, doctors in Taiwan have already effected a cure in more than a million cases among some two million school children who have the disease.

Prospects for a more effective control of leprosy have improved following the discovery of the effectiveness of the sulfone drugs. But many problems remain to be solved in the fight against this disease. They include: the determination of the optimum drug preparation; the development of simple case-finding methods and diagnostic techniques; the establishment of criteria of infectivity; and the need to overcome the centuries-old fear of the disease. WHO is developing its leprosy programs with these objectives in view.

However, long and short term campaigns to combat specific disease, even though successful, may come to nothing if there is not an efficient public health organization within countries to maintain improvements. WHO's most important function is to provide advisory services that help countries strengthen and improve their health services by attacking specific diseases and training professional medical personnel.

In a healthy community like most in North America, ninety-seven children out of every hundred born, grow up to be adults. But in some areas of the world, as many as thirty out of every hundred children die before reaching maturity. The struggle to survive amid poverty and overcrowding is still the rule for most of the world's one billion children. In the area of nutrition, kwashiorkor and other conditions associated with insufficient intake of proteins are of major concern to WHO. Progress has been made in dealing with protein deficiency, especially in the diet of young children after weaning by developing protein-rich foods from local food supplies particularly in countries where milk and milk-products are not generally available. WHO has also made grants to nutrition research centers in India, Uganda, and Guatemala, and considerable advances have resulted from the work undertaken.

The American physician is affected in many ways by the work done by WHO. Examples of international co-operation that directly benefits the physician are: the adoption of international standards for biological substances; establishment of specifications and the selection of nonproprietary names for the most important pharmaceutical preparations; epidemiological intelligence; control of air, land, and sea traffic from the point of view of health; international co-ordination of research; fostering and study of comparable health statistics; world-wide control of habit-forming drugs; and the exchange of scientific information.

Although each nation is responsible for establishing its own specifications for therapeutic agents and medical appliances, this cannot be done without regard to facilities that exist in other countries.

The third World Health Assembly in 1950 approved the publication of the International Pharmacopoeia, a publication that indirectly affects the work of every medical practitioner. This is particularly true when physicians are called to work outside their country, to attend patients coming from other countries, or to share in the aid given to disaster zones when the collaboration of several nations becomes necessary.

Establishment of standards for biological products is another accomplishment of WHO. Standard samples of seventy-five vaccines, antibiotics and other biological preparations have been prepared and are available to laboratories and commercial firms for comparison with their own products.

The ever-increasing international traffic has also raised problems in international health control. Countries which formerly were separated by weeks and even months of travel from areas where disease such as smallpox, yellow fever, cholera, and plague are endemic are today within easy reach by air. For example, international travelers in 1956 were responsible for outbreaks of smallpox in eighteen countries. In eight countries, Ceylon, Ghana, Great Britain, Iran, Italy, Lebanon, Sierra Leone, and Sudan, the outbreaks reached epidemic proportions.

One of the most important achievements of WHO was the adoption of the International Sanitary Regulations in 1952. The regulations apply to sea, land, and air traffic and afford maximum protection against international spread of disease, while assuring the minimum of interference.

Epidemiological intelligence is another example of what can be achieved through international co-operation. Where new outbreaks of quarantinable diseases occur, governments communicate regularly to WHO by cable. This information is transmitted by a network of several radio stations at WHO's disposal. Stations in Geneva, Manila, Saigon, and Djakarta, transmit daily bulletins, while those in Alexandria, Tananarive, Karachi, Madras, Colombo, Hong Kong, Tokyo, Sandakan, and Singapore transmit epidemiological information twice a week.

Although WHO does not itself undertake research, it promotes international collaboration of research. An example of WHO work in this area is the World Influenza Center in London and its network of sixty national influenza laboratory centers located in forty countries. When an outbreak of influenza occurs, the center in the area concerned reports the extent, severity, and identifies the type of virus. The value of this network was strikingly exemplified during the recent world-wide epidemic of "Asian" influenza when a number of countries were able to prepare vaccine before the epidemic reached them.

International health legislation adopted by WHO, which provides uniform registration of diseases and deaths in all countries, is another example of international co-operation. Reliable health statistics are now available from approximately thirty countries, and the list of diseases, injuries and causes of death includes 999 categories. The medical certificate of cause of death which every physician is called upon to utilize follows the pattern of the international form. Thus, every physician participates constantly in the compilation of the mass statistics which are used in etiological and pathological studies.

The main factor behind this progress lies undoubtedly in the good will and the mutual respect WHO has been able to win in the last ten years. Today WHO can count on manpower, resources, and co-operation of universities, institutions, and research centers in all parts of the world.

A number of Minnesotans served on various planning committees for the eleventh annual World Health Assembly. Dr. Charles W. Mayo, Mayo Clinic, Rochester, was chairman of the Minnesota WHO and Centennial Health Committee. Members of the steering committee included: Dr. Robert N. Barr, secretary and executive officer of the Minnesota Department of Health, chairman; Mr. Ray Amberg, director, University of Minnesota Hospitals; Dr. Gaylord W. Anderson, director, University of Minnesota's school of public health; Dr. C. V. E. Cassel, secretary, Minnesota State Dental Association; Mr. Thomas P. Cook, executive secretary, Hennepin County Medical Society; Miss Ragna Gynild, executive secretary, Minnesota Nurses Association; Dr. Benjamin W. Mandelstam, administrator, Mt. Sinai Hospital; Mr. Henry Moen, executive secretary, Minnesota State Pharmaceutical Association; Mr. R. R. Rosell, executive secretary, Minnesota State Medical Association. Medical and health personnel who served on the finance committee were: Dr. Horatio B. Sweetser, president, Minnesota State Medical Association; Mr. Jay Phillips, chairman, board of governors, Mt. Sinai Hospital; Dr. Karl W. Anderson, treasurer, Minnesota State Medical Association.

BLUE SHIELD-BLUE CROSS

Blue Shield professional relations personnel are often asked by physicians the distinction between group and non-group contracts and the reasons for the existence of both. Here are some of the answers.

The majority of Minnesota Blue Shield participant subscribers are enrolled through groups of five or more employes of any firm, agency, or industry. Group enrollment is desirable because each group is likely to include young, middle-aged, and older subscribers, as well as single persons and individuals with family dependents, which generally results in a balance between low risk and high risk individuals. Another important factor is that a member of the group, a group leader, conducts some of the administrative details for the group. There is no individual billing involved by Blue Shield as is the case with non-group subscribers. Thus, the cost of administering the group contract is somewhat less than that experienced in administration of a non-group contract.

Non-group contracts are available to any citizens of Minnesota who are in good health and are less than sixty-five years of age, providing that Blue Shield enrollment is not available on a group basis.

The rates of non-group coverage are slightly higher than the rates for group coverage. The items which make up additional administrative costs in non-group enrollment include processing of application forms, quarterly billing, and accounting costs. In addition, non-group subscribers have a higher average age than do group subscribers in that many non-group applicants are persons close to retirement age who obtain Blue Shield coverage because it is not canceled upon retirement.

In the case of the non-group applicant, a health statement is required. If the health statement from the subscriber is incomplete, it may be necssary to contact the applicant's physician for accurate medical information. If a non-group applicant has received medical or surgical care for some condition prior to application, should the diagnosis of the condition treated warrant it, a rider is attached to his contract excluding benefits for care of that illness, disease, or condition in order to avoid the liability of a known hazard. In view of only a nine month waiting period in the Blue Shield contract for pre-existing conditions, these riders are important from an actuarial viewpoint.

Both the group and non-group contracts are needed to make Blue Shield coverage available to all who desire it, whether or not they are members of a group.

More than 1,140,000 Minnesotans are protected by Blue Cross coverage as of April 30, 1958. The number of Blue Cross contracts in effect as of this date totals 431,769. Compared to the 1,111,140 persons protected and 421,486 contracts in effect as of April 30, 1957, Blue Cross current enrollment represents an increase of 2.6 per cent in number of persons covered and 2.4 per cent in number of contracts in effect.

The month of April, 1958, was a peak month for Blue Cross utilization. Payments to hospitals for subscribers' care reached the all-time high of \$3,010,136. The 19,292 hospital cases paid represented 543 cases per year per 1,000 contracts protected. The 119,027 days of hospital care provided represented 3,352 days of hospital care per year per 1,000 contracts. This was an all-time high in frequency of subscriber usage of Blue Cross benefits.

During the first four months of 1958, the amount of \$11,248,742 was paid to hospitals for the care of Blue Cross subscribers compared to \$9,367,112 during the same period of the previous year. The major factor in the increase of Blue Cross costs for hospital care incurred by subscribers was the increased subscriber usage of Blue Cross benefits. To date this year, 456,379 days of hospital care have been provided 72,259 subscribers. Compared to the same period of the previous year, this represented an increase of 6.7 per cent.

MEDICAL ECONOMICS

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prepayment plans, or a pay increase adequate for such personnel to purchase voluntary prepaid insurance." Texas physicians believe this is a notable history-making step and that it will be emulated by more medical associations as they come to realize that acceptance by the medical profession of any form of government-controlled medical care—no matter how small—is an open invitation for the government to step in and take over the entire practice of medicine, leading to the deterioration of medical care and to the regimentation of physicians and their patients.

MINNESOTA STATE MEDICAL ASSOCIATION

ROSTER—1958

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W. G. Nuessle.......Springfield

CARLTON COUNTY

M. S. Munson.......Barnum
R. H. Puumala.......Cloquet
Alvan Sach-Rowitz.......Moose Lake

CARVER COUNTY

G. T. Schimelpfenig.......Chaska
Leighton Larson.......Waconia
John Clarke.......Watertown

CASS COUNTY

O. F. Ringle.......Walker
C. H. Coombs.......Cass Lake

CHIPPEWA COUNTY

H. A. Roust.......Montevideo

CHISAGO COUNTY

J. E. Halpin.......Rush City

CLAY COUNTY

J. W. Duncan.......Moorhead
V. D. Thysell.......Hawley

CLEARWATER COUNTY

W. E. Anderson.......Clearbrook

COOK COUNTY

W. R. Smith.......Grand Marais

COTTONWOOD COUNTY

H. C. Stratte.......Windom
E. S. Schutz.......Mountain Lake
J. V. Carlson.......Westbrook

CROW WING COUNTY

V. E. Quanstrom.......Brainerd
G. I. Badeaux.......Brainerd
J. B. Nixon.......Crosby

DAKOTA COUNTY

Bernice Thoreson.......South St. Paul
H. J. Just.......Hastings
A. H. Field.......Farmington

DODGE COUNTY

C. E. Bigelow.......Dodge Center
D. E. Affeldt.......Kasson
G. E. Olson.......West Concord

DOUGLAS COUNTY

E. E. Emerson.......Osakis
G. W. Clifford.......Alexandria
E. R. Sather.......Alexandria

FARIBAULT COUNTY

W. C. Chambers.......Blue Earth
M. D. Cooper.......Winnebago
Richard Virnig.......Wells

FILLMORE COUNTY

H. M. Skaug.......Chatfield
L. W. Clark.......Spring Valley
J. P. Nehring.......Preston

FREEBORN COUNTY

S. A. Whitson	Albert Lea
L. E. Steiner	Albert Lea
A. G. Sherman	Albert Lea

GOODHUE COUNTY

R. B. Graves	Red Wing
W. W. Liffrig	Red Wing
G. F. Hartnagel	Red Wing

GRANT COUNTY

L. R. Parson	Elbow Lake
V. A. Doms	Elbow Lake

HENNEPIN COUNTY

Silas C. Andersen	Minneapolis
Bernard I. Saliterman	Minneapolis
Robert D. Semsch	Minneapolis
Arnold S. Anderson	Minneapolis
David W. Feigal	Minneapolis
D. R. Hastings	Minneapolis

HOUSTON COUNTY

N. T. Norris	Caledonia
L. K. Onsgard	Houston
L. A. Knutson	Spring Grove

HUBBARD COUNTY

W. W. Higgs	Park Rapids
John Eiler	Park Rapids

ISANTI COUNTY

W. T. Nygren	Braham
G. E. Larson	Cambridge
J. Pone	Cambridge

ITASCA COUNTY

E. K. Rowles	Coleraine
R. T. Kelly	Grand Rapids
M. J. McKenna	Grand Rapids

JACKSON COUNTY

J. T. Rose	Lakefield
H. A. Christiansen	Jackson
W. H. Halloran	Jackson

KANABEC COUNTY

E. C. Burseth	Mora

KANDIYOHI COUNTY

Howard Bosland	Willmar
R. K. Proeschel	Willmar
Jack Guy	New London

KITTSON COUNTY

J. E. Nord	Hallock

KOOCHICHING COUNTY

R. D. Hanover	International Falls
R. A. MacDonald	Littlefork
C. C. Craig	International Falls

LAC QUI PARLE COUNTY

C. A. Anderson	Madison

LAKE COUNTY

Ralph Papermaster	Two Harbors

LAKE-OF-THE-WOODS

A. A. Brink	Baudette

LINCOLN COUNTY

A. L. Vadheim	Tyler
P. E. Hermanson	Hendricks
C. P. Johnson	Tyler

LYON COUNTY

J. E. Murphy	Marshall
B. C. Ford	Marshall
W. G. Workman	Tracy

MARSHALL COUNTY

Carl Holmstrom	Warren

MARTIN COUNTY

R. C. Hunt	Fairmont
C. W. Krause	Fairmont
H. A. Williamson	Fairmont
Anthony Ourada	Fairmont
M. J. Lester	Truman

McLEOD COUNTY

A. M. Jensen	Brownton
E. W. Lippman	Hutchinson
John Smythe	Lester Prairie

MEEKER COUNTY

Harold Wilmot	Litchfield
Lennox Danielson	Litchfield
Fred Schnell	Litchfield

MILLE LACS COUNTY

Wm. F. McManus	Princeton

MORRISON COUNTY

A. M. Watson	Royalton
O. C. Bolstad	Little Falls
M. L. Hansen	Little Falls

MOWER COUNTY

L. F. Twiggs	Austin
J. G. Havens	Austin

MURRAY COUNTY

B. M. Stevenson	Fulda
R. F. Pierson	Slayton
H. D. Patterson	Slayton

NICOLLET-LE SUEUR COUNTY

D. A. Limbeck	Le Sueur
H. L. Huffington	Waterville
R. A. Curtis	Le Center

NOBLES COUNTY

R. K. Minge	Worthington

NORMAN COUNTY

Byron Kinkade	Ada

OLMSTED COUNTY

N. O. Hanson	Rochester
J. E. Verby	Rochester
T. O. Wellner	Rochester

OTTERTAIL COUNTY

C. A. Boline	Battle Lake
H. K. Helseth	Fergus Falls
C. W. Lewis	Henning
Ralph Estrem	Fergus Falls

PENNINGTON COUNTY

M. D. Starekow	Thief River Falls

PINE COUNTY

A. K. Stratte	Pine City

PIPESTONE COUNTY

W. G. Benjamin	Pipestone
J. G. Lohmann	Pipestone
G. Beckering	Edgerton

POLK COUNTY

C. G. Uhley	Crookston

POPE COUNTY

Paul Swedenburg	Glenwood

RAMSEY COUNTY

A. E. Muller	North St. Paul
O. O. Rollie	Saint Paul

RED LAKE COUNTY

Lester Dale	Red Lake Falls

REDWOOD COUNTY

A. W. Diessner	Redwood Falls
W. E. Johnson	Morgan
S. F. Ceplecha	Redwood Falls

RENVILLE COUNTY

J. A. Cosgriff, Jr.	Olivia
C. A. Anderson	Hector
H. Hinderaker	Bird Island

RICE COUNTY

A. W. Nuetzman	Faribault
Bernard Street	Northfield
Paul G. Bauer	Faribault

ROCK COUNTY

C. L. Sherman	Luverne
A. C. Martin	Luverne
F. W. Bofenkamp	Luverne
E. S. Boone	Luverne
D. M. Odland	Luverne

ROSEAU COUNTY

Lloyd Klefstad	Greenbush

ST. LOUIS COUNTY

H. E. Bakkila	Duluth
H. O. Hoff	Duluth
B. F. Flynn	Hibbing

SCOTT COUNTY

H. M. Juergens	Belle Plaine
Harry N. Simmonds	Prior Lake
J. E. Ponterio	Shakopee

SHERBURNE COUNTY

C. W. Zinn	Elk River

SIBLEY COUNTY

Rolf Hovde	Winthrop
R. H. Kath	Arlington
D. C. Olson	Gaylord

STEARNS-BENTON COUNTY

Henry M. Broker	St. Cloud

STEELE COUNTY

F. C. Anderson	Owatonna
A. J. Floersch	Owatonna
T. W. Stransky	Owatonna

STEVENS COUNTY

O. A. Eide	Hancock

SWIFT COUNTY

R. P. Griffin	Benson
Karl L. Helwig	Kerkhoven
E. J. Kaufman	Appleton

TODD COUNTY

M. E. Mosby	Long Prairie
J. M. Cook	Staples
F. N. Grose	Clarissa

TRAVERSE COUNTY

A. L. Lindberg	Wheaton

WABASHA COUNTY

B. J. Bouquet	Wabasha
W. P. Gjerde	Lake City
D. G. Mahle	Plainview

WADENA COUNTY

L. T. Davis	Wadena
C. H. Pierce	Wadena
W. E. Parker	Sebeka

WASECA COUNTY

S. T. Normann	Waseca
B. J. Gallagher	Waseca

WASHINGTON COUNTY

M. Juergens	Stillwater
C. H. Sherman	Bayport
R. E. Carlson	Stillwater

WATONWAN COUNTY

A. D. Mattson	Saint James
Herbert Boysen	Madelia

WILKIN COUNTY

L. O'Brien	Breckenridge
C. W. Jacobson	Breckenridge

WINONA COUNTY

Herbert vR. Heise	Winona

WRIGHT COUNTY

S. J. Raetz	Maple Lake
R. M. Sandeen	Buffalo

YELLOW MEDICINE COUNTY

Carl Lundell	Granite Falls

Woman's Auxiliary to the Minnesota State Medical Society

Officers

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Mrs. Manley F. Fellows	*President-elect*	Duluth
Mrs. Howard F. Polley	*First Vice President*	Rochester
Mrs. Robert L. Pedersen	*Second Vice President*	Brainerd
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Mrs. John S. Milton	*Corresponding Secretary*	Minneapolis
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Mrs. Fred C. Westerman	*Auditor*	Montgomery
Mrs. John Dordal	*Historian*	Sacred Heart
Mrs. Leo W. Fink	*Parliamentarian*	Minneapolis

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Mrs. David J. Halpern	*Second District*	Brewster
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Mrs. Carl E. Carlson	*Eighth District*	Alexandria
Mrs. Andrew V. Grinley	*Ninth District*	Grand Rapids

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A.M.E.F.—Mrs. John W. Gridley..........Arlington
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Bulletin—Mrs. Milton M. Howell..........Glencoe
Cancer—Mrs. Grant R. Diessner..........Rochester
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Mrs. Conrad J. Holmberg..Minneapolis
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Mrs. Philip Arzt..........Saint Paul
(Minnesota Medicine)—
Mrs. John Linner..........Minneapolis
Finance—Mrs. Charles Froats..........Saint Paul
In Memoriam—Mrs. A. M. Jensen..........Brownton
Legislation—Mrs. L. Raymond Scherer....Minneapolis
Mrs. L. R. Boies..........Minneapolis
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Mrs. Charles Merkert..........Minneapolis
Printing and Roster—Mrs. Karl Anderson....Excelsior
Program Co-ordinator—
Mrs. Howard F. Polley..........Rochester
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Mrs. A. W. Diessner..........Redwood Falls
Mrs. Harold Peterson..........Saint Paul
Mrs. M. O. Wallace..........Duluth
Mrs. I. T. Clark..........Duluth
Today's Health—Mrs. H. P. Van Cleve..........Austin

County Society Roster

Key to Symbols:
***Deceased; †Affiliate, Associate, Resident or Life Members; ‡In Service; ¶Honorary;**
§Wife is Member of Woman's Auxiliary

BLUE EARTH COUNTY MEDICAL SOCIETY

Blue Earth County

Regular meetings, last Monday of the month. Annual meeting, last Monday of December
Number of Members—61

President
VON DRASEK, JOSEPH........Mankato
Secretary
SWENSON, DONALD B........Mankato

§ Anderson, James J........Mankato
Anderson, Margaret C........Mankato
§†Andrews, Roy N........Mankato
§ Batdorf, B. Niles........Mankato
†§Butzer, John A........Mankato
§ Butzer, John F........Mankato
§ Chalgren, William S........Mankato
§ Conley, Robert H........Mankato
Dobson, Mervin W........Mankato
§ Engstrom, Robert B........Mankato
§ Eustermann, John J........Mankato
§ Fortier, Rene G........Mankato
†§Franchere, Frederick Wm.....Lake Crystal
*†§Fugina, George E........Mankato
§ Geurs, Benjamin R........Mankato
§ Gislason, Paul H........Mankato
§ Haes, Julius E........Mankato
§ Hammar, Lawrence M........Mankato
Hankerson, Robert G.....Minnesota Lake
† Hassett, Roger G........Mankato
§ Heimark, John J........Mankato
§ Heller, Edgar E........Mankato
§ Hoeper, Philip G........Mankato
§ Howard, Marshall I........Mankato
† Huffington, Herbert L........Lutsen
§ Jones, Orville H........Mankato
† Juliar, Richard O.....Los Angeles, Calif.
§ Kaufman, Walter B........Mankato
§ Kearney, Rochfort W........Mankato
§†Kemp, Alphonse F........Mankato
†§Koenigsberger, Charles G........Mankato
† Liedloff, Adolph G........Mankato
§ Lindblom, Alton E........No. Mankato
† Luck, Hilda........Mankato
§ McNear, George R., Jr........Mankato
§ Mickelson, John C........Mankato
† Miller, Victor I........Mankato
§ Morgan, Hugh O........Amboy
§ Olive, John T., Jr........Mankato
§ Penn, George E........Mankato
§ Roth, Frederick D........Mankato
Runquist, Richard K.....Good Thunder
§ Sanford, Raymond A........Mankato
§ Scheidel, Alois M........Mankato
† Schmidt, Paul A........Aurora, Illinois
§ Schmitz, Anthony A........Mankato
§ Sjoding, J. Donald........Mankato
§ Smith, Harry J........Lake Crystal
§ Smith, Paul M........Lake Crystal
§ Snider, Howard R........Mankato
§ Stillwell, Walter C........Mankato
§ Swenson, Donald B........Mankato
§ Thiem, Chester E........Mankato
§ Troost, Henry B........Mankato
§ Vezina, John C........Mapleton
§ Von Drasek, Joseph........Mankato
†§Wentworth, Albert J........Mankato
† Williams, Hugh O........Lake Crystal
§ Wohlrabe, John C........No. Mankato
§*Zee, Urban H........Mankato

BLUE EARTH VALLEY MEDICAL SOCIETY

Martin, Watonwan and Faribault Counties

Regular meetings, third Thursday of each month. Annual meeting in November
Number of members—48

President
VIRNIG, RICHARD P........Wells
Secretary
BOYSEN, HERBERT........Madelia

§ Anderson, John W........Blue Earth
§ Armstrong, Ralph S........Winnebago
§ Barr, James S........Elmore
§ Bergman, Oscar B........St. James
§ Boysen, Herbert........Madelia
§ Bratrude, Earl J........St. James
† Chambers, Winslow C........Blue Earth
† Cooper, Maurice D........Winnebago
§ Coulter, Harold E........Madelia
§ Drexler, George W........Blue Earth
§ Gamm, Edgar R........Triumph
§ Gardner, Jack K........Ceylon
§ Gardner, Victor H........Fairmont
§ Halverson, Donald E........Winnebago
§ Halverson, William G........Madelia
§ Hanson, Lewis........Frost
§ Heimark, Julius J........Fairmont
§ Hruza, William J........Madelia
§†Hunt, Roscoe C........Fairmont
§ Kraemer, George N........Fairmont
§ Krause, Carl W........Fairmont
§ Lester, Malcolm J., Jr........Truman
§ Lindahl, Merlyn J........Sherburn
Louisell, Charles T........Fairmont
† McGroarty, John J........Easton
§ Mattson, Albert D........St. James
Mills, John J........Winnebago
§ Moulton, Keith B........St. James
§ Nickerson, John R........Fairmont
§ Nickerson, Neil D........Fairmont
§ Ourada, Anthony L........Fairmont
§ Parsons, R. A........St. James
Parsons, Ralph L........Monterey
† Rollins, Troy G........Rochester
† Rovelstad, Roger A........Yonkers, N. Y.
§ Russ, Homer H........Blue Earth
§ Schulz, Robert W........Fairmont
§ Smith, Don V........Blue Earth
§ Snyder, Clifford D........Keister
§ Thayer, Ellsworth A........Fairmont
§ Vaughn, Victor A........Truman
§ Virnig, Mark P........Wells
§ Virnig, Richard P........Wells
§ Wandke, Otto E........Fairmont
§ Watkins, John A........Wells
§ Williamson, Harold A........Fairmont
† Wilson, Clyde E........Blue Earth
§ Zemke, Erhart E........Fairmont

BROWN COUNTY MEDICAL SOCIETY

Brown County

Regular meetings, quarterly. Annual meeting in January
Number of members—28

President
SCHROEPPEL, JOHN E........Winthrop
Secretary
KAISER, MILTON L........New Ulm

§ Black, William A........New Ulm
§ Burnett, Joseph W........New Ulm
§ Cairns, Robert J........Redwood Falls
§ Carthey, Frank J........New Ulm
§ Dysterheft, Adolf F........Gaylord
§ Fesenmaier, Otto B........New Ulm
§ Fritsche, Albert........New Ulm
§ Fritsche, Carl J........New Ulm
§ Fritsche, Theodore R........New Ulm
Goblirsch, Andrew P........Sleepy Eye
Hovde, Rolf........Winthrop
§ Kaiser, Milton L........New Ulm
§ Keithahn, Elmer E........Sleepy Eye
§ Kitzberger, Peter J........New Ulm
§ Kruzich, Stephen J........Sleepy Eye
†§Kusske, Arthur L........New Ulm
§ Muesing, William J........New Ulm
Nuessle, Walter G........Springfield
§ Penk, Engward L........Springfield
Peterson, Roy A........Vesta
*†Reineke, George F........New Ulm
Rimas, Matthew J........Comfrey
Roan, Morton........Lamberton
§ Saffert, Cornelius A........New Ulm
§ Schroeppel, John E........Winthrop
§ Seifert, Otto J........New Ulm
§ Vogel, Howard A. L........New Ulm
Wohlrabe, Edwin J........Springfield

CAMP RELEASE DISTRICT MEDICAL SOCIETY

Chippewa, Lac qui Parle and Yellow Medicine Counties

Regular meetings, 2nd Thursday each month except July and August

Annual meeting in December

Number of Members—35

President
SCHMIDT, PAUL G. .... Granite Falls
Secretary
ODLAND, OLIN M. .... Granite Falls
§ Allen, John H. .... Montevideo
Anderson, Chester A. .... Madison
§ Barr, Ronald W. .... Montevideo
§ Boody, George J., Jr. .... Sandstone
§ Burns, Floyd M. .... Milan
§ Burns, M. Alpheus .... Milan
‡ Callewart, Robert A. .... Lackland Air Force Base, Texas
§ Camp, Ray Junior .... Madison
Fallon, Virgil T. .... Dawson
† Flom, Robert S. .... Minneapolis
* Guilbert, G. D. .... Minneapolis
§ Hauge, Malvin I. .... Clarkfield
† Holmberg, LeRoy J. .... Canby
§ Hudec, Elwyn R. .... Echo
Hustad, Edward G. .... Montevideo
§ Johnson, Vilhelm M. .... Dawson
† Jordan, Kathleen B. Smith .... Granite Falls
Jordan, Lewis S. .... Granite Falls
§ Kaufman, William C. .... Appleton
† Krystosek, Lee A. .... Madison, Wisc.
Larson, Arthur N. .... Minneapolis
Larson, Dorette W. .... Minneapolis
§ Lima, Ludvig R., Jr. .... Montevideo
§ Lundell, Carl L. .... Granite Falls
§ Maus, Philip .... Dawson
§ Miller, William P. .... Montevideo
Nelson, Melvin S. .... Granite Falls
Niazi, Suad A. .... Clarkfield
§ Odland, Olin M. .... Granite Falls
§ Owens, William A. .... Montevideo
Pertl, Albert L. .... Canby
§ Roust, Henry A. .... Montevideo
§ Schmidt, Paul G., Jr. .... Granite Falls
* Westby, Magnus .... Madison
§ Westby, Norval M. .... Madison

CLAY-BECKER COUNTY MEDICAL SOCIETY

Clay and Becker Counties

Regular meetings, spring and fall. Annual meeting, last week in November

Number of members—28

President
LORENTZEN, ERNEST S. .... Detroit Lakes
Secretary
OLIVER JAMES .... Moorhead
§ Bigler, Earl E. .... Perham
Bigler, Ivan E. .... Perham
§ Bottolfson, Bottolf T. .... Moorhead
§ Carlson, Vernon J. .... Moorhead
§ Dodds, William C. .... Detroit Lakes
§ Duncan, James W. .... Moorhead
§ Geib, Marvin J. .... Fargo, N. Dak.
† Hagen, Olaf J. .... Moorhead
§ Houglum, Arvid J. .... Lake Park
§ Humphrey, Edward W. .... Moorhead
Johnson, Olga H. .... Moorhead
Koons, W. R. .... Mahnomen
§ Larson, Arnold .... Detroit Lakes
§ Lorentzen, Ernest S. .... Detroit Lakes
§ Midthune, Andreen S. .... Lake Park
§ Moberg, Clarence W. .... Detroit Lakes
§ Odland, Mark E. .... Detroit Lakes
Oliver, James .... Moorhead
† Otto, Henry C. .... Frazee
§ Rice, Hagbart G. .... Moorhead
§ Rutledge, John B. .... Detroit Lakes
§ Rutledge, Lloyd H. .... Detroit Lakes
Saxman, Gertrude .... Georgetown
§ Simison, Carl .... Barnesville
† Thysell, Fred A. .... Moorhead
§ Thysell, Vernon D. .... Hawley
§ Watson, Virgil A. .... Detroit Lakes
Watson, Robert H. .... Detroit Lakes

EAST CENTRAL MINNESOTA MEDICAL SOCIETY

Anoka, Chisago, Isanti, Kanabec, Mille Lacs, Pine and Sherburne Counties

Regular meetings, first Tuesday of every other month of the year.

Annual meeting, in December

Number of Members—44

President
HEDENSTROM, PAUL H. .... Cambridge
Secretary
TESCH, GORDON H. .... Elk River
§ Adkins, Galen H. .... Sandstone
Ahlstrom, Robert C. .... Braham
§ Albrecht, Harold H. .... Lindstrom
§ Berge, Harry L. .... Mora
† Bossert, Clarence S. .... Mora
§ Burseth, Edgar C. .... Mora
Corteau, Robert D. .... Onamia
Davis, William I. .... Sandstone
† Dredge, Homer P. .... Minneaoplis
Fryling, Vera B. .... Agnew, California
Gailitis, Veronika M. .... Cambridge
Gully, Raymond J. .... Fort Meade, So. Dak.
Halpin, Joseph E. .... Rush City
§ Hedenstrom, Paul H. .... Cambridge
Henry, Harold W. .... Hinckley
Holmes, Alva E. .... Rush City
§ Hubin, Edwin G. .... Sandstone
Johnson, Aldridge F. .... Isle
Kapsner, Alfred T. .... Princeton
† Kelsey, Carleton G. .... St. Paul
§ Larson, Gerald E. .... Cambridge
§ McManus, William F. .... Princeton
Mach, Ralph F. .... Pine City
§ Magnuson, Raymond C. .... Cambridge
Metcalf, Norman B. .... Princeton
Nelson, Luther A. .... Rush City
§ Nordman, Willard F. .... Mora
Nygren, William T. .... Braham
Peterson, Alvin C. .... Mora
Peterson, Donald B. .... Anoka
Pone, John .... Cambridge
§ Roehlke, Arthur B. .... Elk River
Schut, John W. .... Anoka
Schwartz, Carl A. .... Anoka
§ Spurzem, Raymond J. .... Anoka
§ Stratte, Alf K. .... Pine City
§ Swenson, Roy G. .... North Branch
§ Tesch, Gordon H. .... Elk River
Textor, Jerome .... Anoka
Vik, Melvin .... Cambridge
§ Wadsworth, George L. .... Cambridge
† Waller, Joseph D. .... Pine City
† Woyda, William C. .... Minneapolis
§ Zinn, Charles W. .... Elk River

FREEBORN COUNTY MEDICAL SOCIETY

Freeborn County

Regular meetings, third Thursday of even months. Annual meeting, December

Number of Members—35

President
KEIL, MARCUS A. .... Albert Lea
Secretary
STEINER, LEON E. .... Albert Lea
§ Barr, Lowell C. .... Albert Lea
§ Bartness, John .... Albert Lea
† Burns, Catherine .... Albert Lea
§ Butturff, Carl R. .... Freeborn
†§Calhoun, Frank W. .... Albert Lea
§ Demo, Robert A. .... Albert Lea
§ Donovan, Daniel L. .... Albert Lea
§ Egge, Sanford G. .... Albert Lea
§ Ellertson, Leonard M. .... Albert Lea
§ Erdal, Ove A. .... Albert Lea
§ Folken, Frank G. .... Albert Lea
† Freeman, John P. .... Glenville
§ Gamble, Elbert J. .... Albert Lea
§ Gill, Theodore .... Albert Lea
† Gullickson, Andrew .... Minneapolis
§ Hansen, Theodore M. .... Albert Lea
§ Holian, Darwin K. .... Albert Lea
† Kaasa, Lawrence J. .... Albert Lea
§ Keil, Marcus A. .... Albert Lea
† Leopard, Brand A. .... Brownsville, Texas
§ Menefee, Edward C. .... Albert Lea
§ Neel, Harry B. .... Albert Lea
Nelson, Charles H. .... Albert Lea
§ Nelson, Clayton E. .... Albert Lea
Nelson, Wilhma B. .... Albert Lea
§ Nesheim, Martin O. .... Emmons
§ Palmer, Clinton F. .... Albert Lea
§ Person, John P. .... Albert Lea
§ Prins, Leo R. .... Albert Lea
§ Schmidt, Ruben F. .... Alden
† Schultz, J. Albert .... Albert Lea
§ Sherman, Alfred G. .... Albert Lea
§ Steiner, Leon E. .... Albert Lea
§ Whitson, Sidney A. .... Albert Lea
§ Wilcox, G. Charles .... Albert Lea

GOODHUE COUNTY MEDICAL SOCIETY

Goodhue County
Regular meetings, 2nd Thursday
Annual meeting, December
Number of Members—25

President
Hawley, George M. B., II .... Red Wing
Secretary
Wasmund, Clarence W. .... Red Wing

† Aanes, Almer M. .... Red Wing
§ Ahern, Gene J. .... Red Wing
§ Akins, Willard M. .... Red Wing
§ Billings, Harry H. .... Red Wing
‡ Boswell, J. Thornton .... Kenosha, Wisc.
§ Brusegard, James F. .... Red Wing
‡ Dovenmuehle, Robert H... Durham, N. C.
§ Falls, John L. .... Red Wing
§ Graves, Richard B. .... Red Wing
§ Halvorson, James W. .... Zumbrota
§ Hamilton, Samuel L. .... Red Wing
§ Hartnagel, Grant F. .... Red Wing
§ Hawley, George M. B., II .... Red Wing
*†Jones, Alvah W. .... Red Wing
§ Juers, Edward H. .... Red Wing
§ Larson, Oliver E. H. .... Zumbrota
§ Liffrig, William W. .... Red Wing
§ Miller, Winston R. .... Red Wing
§ Molenaar, Robert E. .... Cannon Falls
§ Sherman, Royal V. .... Red Wing
† Smith, Myron W. .... Red Wing
§ Steffens, Leon A. .... Red Wing
§ Walter, William E. .... Wanamingo
§ Wasmund, Clarence W. .... Red Wing
§ Williams, Marland R. .... Cannon Falls

HENNEPIN COUNTY MEDICAL SOCIETY

Hennepin County
Regular meetings, first Monday of each month.
Annual meeting, first Monday in October
Number of Members—1,081

President
Nelson, O. L. Norman .... Minneapolis
Secretary
Holmberg, Conrad J. .... Minneapolis
Exec. Sec'y
Cook, Thomas P. .... Minneapolis

Abramson, Milton .... Minneapolis
§ Adkins, Charles D. .... Minneapolis
§ Agustsson, Hreidar .... Minneapolis
§ Ahern, Eugene E. .... Minneapolis
§ Alexander, Harlan A. .... Minneapolis
§ Aling, Charles A. .... Minneapolis
† Altnow, Hugo O. .... Coral Gables, Fla.
§ Amatuzio, Donald S. .... Minneapolis
§ Andersen, Silas C. .... Minneapolis
§ Anderson, Arnold S. .... St. Louis Park
§ Anderson, David M. .... St. Louis Park
† Anderson, Edward D. .... Gstaad, Switzerland
§ Anderson, Ernest R. .... Minneapolis
§ Anderson, Frank J. .... Minneapolis
Anderson, John A. .... Minneapolis
§ Anderson, John T. .... Minneapolis
§ Anderson, Karl W. .... Minneapolis
§ Anderson, Roger L. .... Minneapolis
§ Anderson, U. Schuyler .... Minneapolis
Anderson, Wallace E. .... Minneapolis
§ Anderson, William H. .... Minneapolis
Anderson, William T. .... Minneapolis
Andreassen, Einar C. .... St. Paul
§ Andreassen, Rolf L. .... Minneapolis
§ Andresen, Karl d'A. .... Minneapolis
§ Andrews, Robert S. .... Minneapolis
§ Ankner, Frank J. .... Minneapolis
§ Anonsen, Richard E. .... Minneapolis
Ansell, Julian S. .... Minneapolis
† Arends, Archabald L. .... Minneapolis
§ Arey, Stuart Lane .... Minneapolis
§ Arhelger, Stuart W. .... Minneapolis
§ Arlander, Clarence E. .... Minneapolis
§ Arling, Leonard S. .... Minneapolis
§ Arms, James J. .... Minneapolis
Armstrong, Byron H. .... Hopkins
Arnold, Ann W. .... Minneapolis
Arvidson, Carl G. .... Minneapolis
*†Aune, Martin .... Minneapolis
§ Baggenstoss, Osmond J. .... Minneapolis
§ Bagley, Russell W. .... Minneapolis
§ Baird, Joseph W. .... Minneapolis
§ Baken, Melvin P. .... Minneapolis
Baker, Abe B. .... Minneapolis
† Baker, Alfred T. .... Minneapolis
§ Baker, Milton E. .... Minneapolis
Bakke, Arnold C. .... Minneapolis
§ Baleisis, Peter .... Minneapolis
§ Balkin, Samuel G. .... Minneapolis
§ Balogh, Charles J. .... Minneapolis
Bank, Harry E. .... Unknown
Barnett, Robert M. .... Minneapolis
§ Barno, Alex .... St. Louis Park
§ Barr, Maxwell M. .... Minneapolis
Barr, Robert N. .... Minneapolis
Barron, Jesse J. .... Minneapolis
†§Barron, Moses .... Minneapolis
Barron, S. Steven .... Minneapolis
§ Beach, Northrop .... Minneapolis
† Becker, Arnetta M. .... Lincoln, Nebraska
§ Bedford, Edgar W. .... Minneapolis
§ Beierstein, Samuel .... Minneapolis
§ Beiswanger, Richard H. .... Minneapolis
† Bell, Elexious T. .... Minneapolis
§ Bellville, Titus P. .... Minneapolis
§ Belzer, Meyer S. .... Minneapolis
Benesh, Louis A. .... Minneapolis
§ Benjamin, Edwin G. .... Minneapolis
§ Benjamin, Harold G. .... Minneapolis
Berg, Clinton C. .... Wayzata
Berger, Alex G. .... Minneapolis
§ Bergh, George S. .... Minneapolis
Bergh, Solveig M. .... Minneapolis
§ Berglund, Eldon B. .... Minneapolis
§ Bergquist, James R. .... Minneapolis
§ Berkwitz, Nathaniel J. .... Minneapolis
Berman, Reuben .... Minneapolis
Bernhisel, Harris G. .... Minneapolis
Bernstein, Irving C. .... Minneapolis
Berris, Harold .... Minneapolis
§ Bessesen, Alfred N., Jr. .... Minneapolis
Bevis, William D. .... Minneapolis
Bieter, Raymond N. .... Minneapolis
§ Bilka, Paul J. .... Minneapolis
§ Binder, Manuel R. .... Minneapolis
§ Blake, Allan J. .... Hopkins
§ Blake, James A. .... Hopkins
§ Blake, Paul S. .... Hopkins
§ Bloedel, Traugott J. .... Osseo
§ Blomberg, Robert D. .... Minneapolis
§ Bloom, Norman B. .... Minneapolis
§ Blumenthal, Jacob S. .... Minneapolis
§ Boehrer, John J., Jr. .... Minneapolis
§ Bofenkamp, Benjamin .... Minneapolis
§ Bohn, Donald G. .... Minneapolis
§ Boies, Lawrence R. .... Minneapolis
† Booth, Albert E. .... Minneapolis
† Boreen, Clifton A. .... Minneapolis
† Borgeson, Egbert J. .... St. Paul
§ Borman, Chauncey N. .... Minneapolis
§ Borowicz, Leonard A. .... Minneapolis
§ Bowers, Gordon G. .... Minneapolis
Boynton, Ruth E. .... Minneapolis
Bradley, Jeanne B. .... Minneapolis
Bradley, John G. .... Minneapolis
Brandt, Henry E. .... Minneapolis
†§Bratrud, Arthur F. .... Minneapolis
Bravick, Donald D. .... Minneapolis
Breitenbucher, Robert B. .... Minneapolis
§ Brekke, Harvey J. .... Minneapolis
§ Bridge, Allyn G. .... Minneapolis
Brill, Alice K. .... Minneapolis
† Brooks, Charles N. .... Minneapolis
† Brown, Edgar D. .... Unknown
Brown, William D. .... Minneapolis
§ Buchstein, Harold F. .... Minneapolis
§ Buirge, Raymond E. .... Minneapolis
† Bulkley, Kenneth .... Minneapolis
§ Burnham, Wesley H. .... Minneapolis
§ Bushard, Wilfred J. .... Minneapolis
§ Buzzelle, Leonard K. .... Minneapolis
§ Cable, Morris L. .... Minneapolis
§ Cabot, Clyde M. .... Minneapolis
§ Cady, Laurence H. .... Minneapolis
Cameron, Isabell L. .... Minneapolis
† Campbell, Lowell M. .... Minneapolis
§ Campbell, Orwood J. .... Minneapolis
§ Caplan, Leslie .... Minneapolis
§ Card, William H. .... Minneapolis
§ Carey, James B. .... Minneapolis
§ Carey, James B., Jr. .... Minneapolis
§ Carlander, Lester W., Jr. .... Minneapolis
Carlson, Charles V. .... Mound
§ Carlson, Lawrence .... Minneapolis
§ Carlson, Leonard T. .... Minneapolis
§ Caron, Robert P. .... Minneapolis
§ Carr, William J. .... Minneapolis
§ Caspers, Carl G. .... Minneapolis
Cauble, Charles F. .... Minneapolis
†§Cavanor, Frank T. .... Minneapolis
§ Ceder, Elmer T. .... Minneapolis
Cella, Joseph A. .... Minneapolis
Chadbourn, Wayne A. .... Minneapolis
Challman, S. Alan .... Minneapolis
† Chavez, Demetrio A. .... St. Paul
§ Chesler, Merrill D. .... Minneapolis
§ Child, Sherman B. .... Minneapolis
§ Chisholm, Tague C. .... Minneapolis
§ Christensen, Llewellyn E. .... Minneapolis
Clark, Malcolm D. .... Minneapolis
§ Clark, Robert S. .... Minneapolis
§ Clay, Lyman B. .... Minneapolis
§ Cochrane, Ray F. .... Minneapolis
Coe, John I. .... Minneapolis
§ Cohen, Bernard A. .... Minneapolis
Cohen, Ephriam B. .... Minneapolis
Cohen, Henry W. .... Minneapolis
† Cohen, Maynard M. .... Minneapolis
§ Cohen, Sumner S. .... Oak Terrace
§ Cole, James S. .... Minneapolis
§ Cooper, John P. .... Minneapolis
§ Cooper, Robert R. .... Minneapolis
Corniea, Albert D. .... Minneapolis
§ Correa, Dale H. .... Minneapolis
Cowan, Donald W. .... Minneapolis
Craig, M. Elizabeth .... Minneapolis
†§Cranmer, Richard R. .... Minneapolis
Cranston, Robert W. .... Minneapolis
§ Creevy, Charles D. .... Minneapolis
§ Culligan, Leo C. .... Minneapolis
§ Cundy, Donald T. .... Minneapolis
Cushing, Richard T. .... St. Louis Park
† Cutts, George .... Minneapolis
*†Dady, Elmer E. .... Minneapolis
§ Daggett, Donald R. .... Minneapolis
§ Dahl, Elmer O. .... Minneapolis
Dahl, James C. .... Minneapolis
§ Dahl, John A. .... Minneapolis
† Daniel, Donald H. .... Minneapolis
§ Danyluk, Michael .... Minneapolis
§ Dargay, Cyril P. .... Minneapolis
§ David, Reuben .... Hopkins
§ Davis, Jay C. .... Minneapolis
§ del Plaine, Carlos W. .... Minneapolis
§ Devereaux, Thomas J. .... Wayzata
† DeWall, Richard A. .... Minneapolis
Dickman, Roy W. .... Minneapolis
§ Diefenbach, Eugene J., Jr. .... Minneapolis
§ Diehl, Harold S. .... New York
§ Dierker, Heinrich A. .... Minneapolis
Diessner, Henry D. .... Minneapolis
§ Doan, Robert E. .... Wayzata
§ Donatelle, Edward P. .... Minneapolis
§ Dorge, Richard I. .... Minneapolis
§ Dorsey, George C. .... Minneapolis
Doscherholmen, Alfred .... Minneapolis
† Doxey, Gilbert L. .... Minneapolis
§ Doyle, Lawrence O. .... Minneapolis
†§Drake, Charles R. .... Minneapolis
§ Drill, Herman E. .... Hopkins
§ Duff, Edwin R. .... Minneapolis
Dummer, Donald J. .... New Brighton
§ Dunlap, Earl H. .... Minneapolis
§ Dupont, Joseph A. .... Excelsior
Duryea, Marbry .... Minneapolis
§ Duryea, Willis M. .... Minneapolis
§ Duryea, Willis M., Jr. .... Minneapolis
§ Dvorak, Benjamin A. .... Minneapolis
§ Dwan, Paul F. .... Minneapolis
§ Dworsky, Samuel D. .... Minneapolis
§ Eder, Walter P. .... Minneapolis
§ Ehrenberg, Claude J. .... Minneapolis

§ Ehrlich, S. Paul	Minneapolis
Eich, Matthew A.	Minneapolis
§ Eichhorn, Edmund P., Jr.	Minneapolis
Eisenstadt, David H.	Minneapolis
§ Eisenstadt, William S.	Minneapolis
Eitel, George D.	Minneapolis
§ Eli, Earl W.	Minneapolis
§ Ellison, David E.	Minneapolis
§ Ellison, Ellis	Minneapolis
Ellison, Evan S.	Minneapolis
Ellwood, Paul M., Jr.	Minneapolis
Elrod, Calvin R.	Long Lake
§ Emond, Albert J.	Farmington
Emond, Joseph S.	Farmington
§ Engel, Joseph P.	Minneapolis
§ Engelhart, Peter C.	Minneapolis
§ Englund, Elvin F.	Minneapolis
§ Engstrand, Oscar J.	Minneapolis
§ Engstrom, Denton P.	Minneapolis
§ Erickson, Clifford O.	Minneapolis
§ Erickson, Laurence F.	Minneapolis
§ Erickson, Myron E.	Minneapolis
§ Erickson, Reuben F.	Minneapolis
Erlanson, Andrew C.	St. Paul
§ Esensten, Sidney	Minneapolis
§ Evans, Edward T.	Minneapolis
Evans, Robert D.	Minneapolis
† Fahr, George E.	Minneapolis
§ Fansler, Walter A.	Minneapolis
§ Feeney, John M.	Minneapolis
§ Feigal, David W.	Wayzata
Feinberg, Philip	Minneapolis
Feinberg, Samuel B.	Minneapolis
§ Feinstein, Julius Y.	Minneapolis
§ Fenger, Ejvind P. K.	Oak Terrace
§ Field, Charles W.	Minneapolis
Fifer, William R.	Minneapolis
§ Fingerman, David L.	Minneapolis
§ Fink, Leo W.	Minneapolis
§ Fink, Robert J.	Minneapolis
§ Fink, Walter H.	Minneapolis
§ Fisher, Isadore I.	Minneapolis
† Fitzgerald, Don F.	Wayzata
†§Fjeldstad, C. Alford	Minneapolis
Fleeson, William H.	Minneapolis
Fleming, Dean S.	Hopkins
§ Fliehr, Richard R.	Minneapolis
Flink, Edmund B.	Minneapolis
Foker, Leslie W.	Minneapolis
§ Folsom, Louis B.	Minneapolis
§ Ford, William H.	Minneapolis
§ Foster, Orley W.	Minneapolis
†§Fowler, L. Haynes	Minneapolis
† Fox, Donald P.	Tanganyika, E. Africa
§ Fox, James Rogers	Minneapolis
§ Frane, Donald B.	Minneapolis
§ Fredericks, George M.	Minneapolis
Freeman, Donald W.	St. Louis Park
‡ Freeman, Craig W.	Minneapolis
§ French, Lyle A.	Minneapolis
§ Frey, Richard J.	Minneapolis
§ Friberg, Joseph B.	Minneapolis
§ Fried, Louis A.	Minneapolis
§ Friedell, Aaron	Minneapolis
Friedell, George	St. Louis Park
Friedlieb, Oskar P.	Minneapolis
§ Friedman, Harry S.	Minneapolis
Friend, Charles A.	Minneapolis
§ Frost, John B.	Minneapolis
§ Frost, Russell H.	Oak Terrace
§ Frykman, Howard M.	Minneapolis
§ Frys, Russell N.	Minneapolis
Fuller, Alice H.	Minneapolis
§ Funk, Victor K.	Oak Terrace
Furman, Lucie C.	Minneapolis
§ Fusaro, Ramon M.	Minneapolis
§ Gaard, Richard C.	Minneapolis
§ Gallett, Lester E.	Minneapolis
† Galligan, Margaret, M.D.	Minneapolis
§ Gammell, John H.	Minneapolis
§ Garamella, Joseph J.	Minneapolis
§ Garske, George L.	Minneapolis
Garten, Joseph L.	Minneapolis
§ Garvey, James T.	Minneapolis
Gault, N. L., Jr.	Minneapolis
Gault, Sarah J.	Minneapolis
§ Gaviser, David	Minneapolis
George, Vane P., Jr.	Minneapolis
§ Gibbs, Robert W.	Minneapolis
§ Giebenhain, John N.	Minneapolis
§ Giere, Joseph C.	Minneapolis
§ Giere, Richard W.	Minneapolis
Gilbert, Maurice G.	Minneapolis
Gingold, Benjamin A.	Minneapolis
† Girvin, Richard B.	Des Peres, Mo.
§ Glaeser, John H.	Minneapolis
§ Glassberg, Irving J.	Minneapolis
§ Glueck, Bernard C., Jr.	Minneapolis
Goetz, Frederick C.	Minneapolis
§ Gold, David	Minneapolis
§ Goldberg, Isadore M.	Minneapolis
Goldberg, Marvin E.	Minneapolis
§ Goldman, Theodore I.	Minneapolis
§ Goldner, Meyer Z.	Minneapolis
§ Goltz, Robert W.	Minneapolis
§ Good, Hoff D.	Minneapolis
Goodchild, William R.	Minneapolis
Gordon, John R.	Minneapolis
Gordon, Philip E.	Minneapolis
§ Gordon, Sewell S.	St. Louis Park
Grant, Suzanne	Minneapolis
†§Gratzek, Frank R.	Minneapolis
Gray, Royal C.	Minneapolis
Green, Clayton Robbie	Excelsior
Green, Robert A.	St. Louis Park
§ Greenberg, Albert J.	Minneapolis
§ Greenfield, Irving	Minneapolis
Greenspan, Richard H.	Minneapolis
¶ Greishiemer, Esther M.	Philadelphia, Pa.
Grimes, Marian	Minneapolis
§ Grimmell, Francis J.	Minneapolis
§ Gronvall, Paul R.	Minneapolis
§ Grotting, John K.	Minneapolis
§ Gullickson, Glenn, Jr.	Minneapolis
§ Gunlaugson, Frederick G.	Minneapolis
§ Gushurst, Edward G.	Palm Springs, Calif.
§ Gustafson, Paul O.	Minneapolis
§ Gustason, Harold T.	Minneapolis
Haberer, Helen R.	Minneapolis
‡ Haberland, Lyle F.	New York
Haberle, Charles A.	Minneapolis
§ Hagen, Kristofer	Minneapolis
§ Hagen, Wayne S.	Minneapolis
† Haggard, George D.	Minneapolis
§ Hall, Harry B.	Minneapolis
§ Hall, Wendell H.	Minneapolis
§ Hamel, Joseph I.	Minneapolis
Hammerstrom, Robert N.	Minneapolis
§ Hannah, Hewitt B.	Minneapolis
§ Hansen, Cyrus O.	Minneapolis
§ Hansen, Erling W.	Minneapolis
Hansen, Olga S.	Minneapolis
§ Hansen, Rollin M.	Minneapolis
Hanske, Edward A.	Minneapolis
§ Hanson, Harlow J.	Minneapolis
Hanson, Harold B.	Minneapolis
† Hanson, Henry V.	St. Paul
§ Hanson, Malcolm B.	Minneapolis
§ Hanson, Mark C. L.	Minneapolis
§ Hanson, William A.	Minneapolis
§ Happe, Lawrence J.	Minneapolis
Harris, Leon D.	Minneapolis
§ Harrison, William C.	Minneapolis
Hartman, Evelyn E.	Minneapolis
§ Hass, Frederick M.	Minneapolis
§ Hastings, DeForest R.	Minneapolis
§ Hastings, Donald W.	Minneapolis
§ Hauge, Erling T.	Minneapolis
§ Haugen, George W.	Minneapolis
Haugen, John A.	Minneapolis
§ Hauser, Donald C.	Minneapolis
Hauser, George W.	Minneapolis
§ Havel, Robert J.	Minneapolis
§ Haven, Walter K.	Minneapolis
§ Hawkinson, Raymond P.	Minneapolis
§ Hay, Lyle J.	Minneapolis
† Hayes, James M.	Arcadia, Calif.
§ Hays, Albert T.	Minneapolis
† Hawya, Eugene W.	Minneapolis
§ Head, Douglas P.	Minneapolis
§ Hebbel, Robert	Minneapolis
† Hendrickson, John F.	Minneapolis
§ Henrikson, Earl C.	Minneapolis
† Henry, Clifford E.	Kirksville, Mo.
Herbert, Willis L.	Minneapolis
§ Hermann, Harold W.	Minneapolis
†§Higgins, John H.	Minneapolis
Hilgermann, George O.	Minneapolis
Hill, Earl	Minneapolis
§ Hill, Elmer M.	Minneapolis
† Hillis, Samuel J.	E. Bradenton, Fla.
Hinckley, Robert G.	Minneapolis
§ Hirschfield, Frank R.	Minneapolis
§ Hitchcock, Claude R.	Minneapolis
§ Hodges, Kenneth V.	Minneapolis
Hoffbauer, Frederick W.	Minneapolis
§ Hoffert, Henry E.	Minneapolis
§ Hoffman, Roy A.	Minneapolis
§ Hoffman, Walter L.	Minneapolis
§ Holmberg, Conrad J.	Minneapolis
§ Holzapfel, Fred C.	Minneapolis
§ Hoppes, Emerson E.	Minneapolis
Horns, Howard L.	Minneapolis
§ Horns, Richard C.	Minneapolis
Houkom, Bjarne	Minneapolis
§ Houle, Rollin J.	New Brighton
Hovland, Melvin L.	Minneapolis
Howard, Robert B.	Minneapolis
Howard, Solomon E.	Minneapolis
§ Howell, Carter W.	Minneapolis
Hudson, George E.	Minneapolis
†§Huenekens, Edgar J.	Minneapolis
Hulteng, Donald B.	Minneapolis
§ Hultkrans, Rudolph E.	Minneapolis
† Hurd, Annah	Minneapolis
Hutchinson, Dorothy W.	Oak Terrace
§ Hymes, Charles	Minneapolis
† Hynes, John E.	Minneapolis
Ide, Arthur W., Jr.	Minneapolis
§ Idstrom, Linneus G.	Minneapolis
§ Indeck, Walter	Minneapolis
§ Ingalls, Edgar G.	Minneapolis
† Irvine, Harry G.	Minneapolis
§ Iverson, Rolf M.	Minneapolis
§ Jacobson, Loren J.	Minneapolis
§ Jacobson, Wyman E.	St. Louis Park
§ Jay, Alan R.	Minneapolis
§ Jefferies, William L.	St. Louis Park
§ Jensen, Harry C.	Minneapolis
† Jensen, Maurius J.	Minneapolis
Jensen, Nathan K.	Minneapolis
Jensen, Reynold A.	Minneapolis
Jepson, William W.	Minneapolis
Jerome, Bourne	Minneapolis
Jerome, Elizabeth K.	Minneapolis
Jeub, Robert P.	Minneapolis
§ Johnson, Angelo G.	Minneapolis
§ Johnson, Arthur B.	Minneapolis
§ Johnson, David R.	Minneapolis
§ Johnson, Frank E.	Minneapolis
§ Johnson, Harry A.	Minneapolis
†§Johnson, James A.	Minneapolis
§ Johnson, John W.	Minneapolis
*†§Johnson, Julius	Minneapolis
§ Johnson, Malcolm R.	Minneapolis
Johnson, Morris B.	Minneapolis
Johnson, Norman P.	Minneapolis
Johnson, Norman Paul	Minneapolis
§ Johnson, Norton T.	Minneapolis
§ Johnson, Paul E.	Minneapolis
§ Johnson, Reinald G.	Minneapolis
§ Johnson, Reuben A.	Minneapolis
§ Johnson, Richard S.	Minneapolis
§ Johnson, Robert E.	Minneapolis
Johnson, Roger S.	Wayzata
Johnson, Youbert T.	Minneapolis
Jones, David G.	Minneapolis
†§Jones, Herbert W., Jr.	Minneapolis
§ Jones, Richard H.	Minneapolis
§ Jones, William R.	Minneapolis
Josewich, Alexander	Minneapolis
§ Judd, Allen S.	Minneapolis
§ Judd, Walter H.	Washington, D. C.
§ Jurdy, Mitchell J.	Minneapolis
§ Kadesky, Harold B.	Minneapolis
§ Kalin, Oscar T.	Minneapolis
§ Kallestad, Leonard L.	Wayzata
§ Kantar, Bruce L.	Minneapolis
§ Kaplan, Harold A.	Minneapolis
§ Kaplan, J. Jacob	Minneapolis
§ Karleen, Conrad I.	Minneapolis
§ Karlen, Markle	Minneapolis
Kasper, Robert E.	Minneapolis
Katz, Yale J.	Los Angeles, Calif.
Kaufman, Herschel J.	Minneapolis
§ Kelby, Gjert M.	Minneapolis
§ Kelly, Charles F.	Minneapolis
§ Kelly, John P.	Minneapolis
Kennedy, Byrl J.	Minneapolis
†§Kennedy, Claude C.	Minneapolis
† Kennedy, Jane F.	Minneapolis
§ Kerkhof, Arthur C.	Minneapolis
Kiesler, Frank, Jr.	Minneapolis
§ Kimmel, George C.	Minneapolis
King, Frances W.	Oak Terrace
§ Kinsella, Thomas J.	Minneapolis
*†Kistler, Alvin J.	Minneapolis
Kjenaas, Ervin A.	Hastings
†§Kline, Richard F.	St. Cloud
§ Knapp, Miland E.	Minneapolis
†§Knight, Ralph T.	Minneapolis
† Knight, Ray R.	Minneapolis
Knudsen, Helen L.	Minneapolis
§ Koepcke, Gerald M.	Minneapolis
Kohlhase, Robert E.	Minneapolis
Koller, Hermann M.	Minneapolis
Koller, Louis R.	Minneapolis
Koller, Robert L.	Hopkins
Korchik, John P.	Minneapolis
Kosiak, John, Jr.	Minneapolis
Kottke, Frederic J.	Minneapolis
§ Koucky, Rudolph W.	Minneapolis
‡§Kovack, Freeman D.	Amarillo, Texas
§ Krafft, Walter E.	Minneapolis
§ Kremen, Arnold J.	Minneapolis
§ Krieser, Albert E.	Minneapolis
§ Kucera, Frank J.	Hopkins
† Kucera, William J.	Santa Barbara, Calif.
Kump, Warren L.	Minneapolis
§ Kusz, Clarence V.	Minneapolis
LaBree, John W.	St. Louis Park
Lagaard, Sheldon M.	Minneapolis
† Lajoie, John M.	Minneapolis
§ Lamb, H. Douglas	Minneapolis
§ Lang, Leonard A.	Minneapolis
†§Lapierre, Arthur P.	Minneapolis
§ Larsen, Frank W.	Minneapolis
§ Larson, Arthur K.	Minneapolis
§ Larson, Clarence M.	Minneapolis
§ Larson, Donald M.	Minneapolis
§ Larson, Lawrence M.	Minneapolis

Larson, Leonard M..................Oak Terrace
§ Larson, Paul N..................Minneapolis
Larson, Roger C..................Minneapolis
Latts, Elliott M..................Minneapolis
† LaVake, Rae T..................Minneapolis
Laymon, Carl W..................Minneapolis
§ Leavenworth, Richard O., Jr..............
Minneapolis
§ Leemhuis, Andrew J. ....Minneapolis
§ Leiferman, Robert J..................Minneapolis
Leinonen, Wendla E..................Anoka
§ Leland, Harold R..................Minneapolis
§ Lenz, O. A..................Minneapolis
§ Leonard, Lawrence J..................Minneapolis
§ Leonard, Samuel..................Minneapolis
Lerner, A. Ross..................Minneapolis
§ Lester, Richard G..................Minneapolis
§ Lewis, Joyce S., Jr..................Minneapolis
Lienke, Roger I..................Minneapolis
Lillehei, C. Walton..................Minneapolis
Lillehei, Elmer J..................Robbinsdale
‡ Lind, Carl J., Jr.....Fort McPherson, Ga.
Lindberg, Arthur N..................Minneapolis
† Lindberg, Arvid C..................Minneapolis
§ Lindberg, Vernon L..................Minneapolis
§ Lindberg, Winston R..................Minneapolis
§ Lindblom, Maurice L..................Minneapolis
§ Lindemann, Charles E..................Minneapolis
§ Linderholm, Bruce E..................Minneapolis
§ Lindgren, Russell C..................Minneapolis
§ Lindquist, Richard H..................Minneapolis
§ Linner, Gunnar..................Binneapolis
†§Linner, Henry P..................Minneapolis
§ Linner, John H..................Minneapolis
§ Linner, Paul W..................Minneapolis
Lippman, Emanuel S..................Minneapolis
§ Lipschultz, Oscar..................Minneapolis
† Litchfield, John T..................Minneapolis
Litman, Abraham B..................Minneapolis
Lober, Paul H..................Minneapolis
§ Lofsness, Stanley V..................St. Paul
†§Logefeil, Rudolph C..................Minneapolis
Logothetis, John A..................Minneapolis
† Loomis, Earl A..................Minneapolis
§ Lott, Frederick H..................Minneapolis
Lovett, Beatrice R..................Oak Terrace
Lowry, Elizabeth C..................Minneapolis
§ Lowry, Paul T..................Minneapolis
Lowry, Thomas..................Minneapolis
Lueck, Wallace W..................Minneapolis
§ Lufkin, Nathaniel H..................Minneapolis
Lund, George W..................Minneapolis
Lundberg, Ruth I..................Minneapolis
Lundblad, Roy A..................Minneapolis
Lundblad, Stanley W..................Minneapolis
§ Lundeberg, Karl R..................Minneapolis
§ Lundquist, Virgil J. P..................Minneapolis
† Lynch, Matthew J..................Minneapolis
§ Lyon, John D., Jr..................Hopkins
§ Lyons, James H..................Minneapolis
Lyons, Michael W..................Minneapolis
†§ Lysne, Henry..................Minneapolis
§ Lysne, Myron..................Robbinsdale
† Lysyj, Anatol ....Minneapolis
§ Lyzenga, Anton G..................Minneapolis

McCaffrey, F. John..................Minneapolis
§ McCampbell, Malcolm D..................Minneapolis
§ McCannel, Malcolm A..................Minneapolis
McCarthy, Donald..................St. Paul
McCartney, James S..................Minneapolis
§ McCormick, Donald P..................Minneapolis
† McDaniel, Orianna..................Minneapolis
§ McFarland, Arthur H..................Minneapolis
§ McGandy, Robert F..................Minneapolis
§ McGeary, George E..................Minneapolis
§ McInerny, Maurice W..................Minneapolis
McKelvey, John L..................Minneapolis
§ McKenzie, Charles H..................Minneapolis
§ McKinlay, Chauncey A. ....Minneapolis
§ McKinlay, Gordon L..................Minneapolis
§ McKinney, Frank S..................Minneapolis
§ McLaughlin, Byron H..................Minneapolis
§ McMurtrie, William B..................Minneapolis
McNeil, John J..................Minneapolis
§ McPheeters, Herman O..................Minneapolis
† McQuarrie, Irvine..................Minneapolis
†§MacDonald, Daniel A..................Minneapolis
§ MacDonald, John W..................Minneapolis
§ MacKinnon, Donald C..................Minneapolis
§ Mach, Frank B..................Minneapolis
Macheledt, Neil L..................Anoka
§ Meader, Edward C..................Minneapolis
Magraw, Richard M..................Minneapolis
Mahle, James P..................Minneapolis
†§Maland, Clarence O..................Minneapolis
Mandel, Sheldon L..................Minneapolis
§ Mankey, James C..................Minneapolis
Mann, George A..................Minneapolis
‡ Margulis, Alexander R..................
Fort Bragg, N. C.
Mark, Aaron L..................Minneapolis
§ Marking, George H. ....Minneapolis
Martin, Frank E..................Minneapolis
§ Martin, George R..................Minneapolis
Martinson, Carl J..................Wayzata
§ Martinson, Elmer J..................Wayzata
*§Mattill, Peter M..................Oak Terrace
§ Mattson, Hamlin A. N..................Minneapolis
Maunder, John B..................Minneapolis
†§Maxeiner, Stanley R..................Minneapolis
§ Maxeiner, Stanley R., Jr..................Minneapolis
§ Meller, Robert L..................Minneapolis
Mendelson, David F..................Minneapolis
§ Merkert, Charles E..................Minneapolis
§ Merkert, George L..................Minneapolis
† Merrick, Charlotte T..................St. Paul
Messenheimer, Myron G..................Minneapolis
§ Meyer, Alvin J..................Minneapolis
† Meyer, E. Lawrence..................Minneapolis
Meyer, Robert J..................Minneapolis
Michael, Joseph C..................Minneapolis
§ Michel, Henry H..................Minneapolis
§ Michelson, Henry E..................Minneapolis
† Mickelsen, Emma F..................Minneapolis
Middlebrook, John E.......... ....Minneapolis
Miller, Arden L..................Minneapolis
§ Miller, Harold E..................Minneapolis
§ Miller, Hugo E..................Minneapolis
§ Miller, John Carleton..................Minneapolis
§ Millett, D. Keith..................Minneapolis
§ Milton, John S..................Minneapolis
Minsky, Armen A..................Minneapolis
§ Mitby, Irvin L..................Minneapolis
§ Mitchell, Berton D..................Minneapolis
Mitchell, Edward C..................Minneapolis
§ Mitchell, Mancel T..................Minneapol'
§ Mixer, Harry W..................Minneapo
§ Moe, John H..................Minneapolis
§ Moehn, John T..................Minneapolis
Moen, Johannes K., Jr..................Minneapolis
† Monahan, Elizabeth S..................Minneapolis
§ Monson, Einer M..................Minneapolis
§ Moore, Irvin H..................Minneapolis
Moorhead, Marie..................Minneapolis
§ Moos, Daniel J..................Minneapolis
§ Mork, A. Harold..................Anoka
§ Mork, Frank E..................Anoka
Morrell, Frank..................Minneapolis
Morrison, Charlotte J..................Minneapolis
§ Mosser, Donn G..................Minneapolis
§ Moyer, Leonard B. ....Minneapolis
§ Mulholland, William M..................Minneapolis
Murphy, Edmund P..................Minneapolis
Murray, Elisabeth M..................Minneapolis
§ Muske, Marvin M..................Minneapolis
§ Musty, Nicholas J..................Minneapolis
§ Myers, Jay A..................Minneapolis
§ Myhre, James..................Minneapolis

Nagobads, Visvaldis J..................Minneapolis
Nash, Eldore B..................Minneapolis
§ Naslund, Ames W..................Minneapolis
Nauth, Bernard S..................Minneapolis
§ Neal, Joe M..................St. Paul
§ Neary, Richard P..................Minneapolis
† Nelson, Bernette G..................Hoyt Lakes
† Nelson, Bernice A..................Minneapolis
Nelson, C. Barton..................Minneapolis
Nelson, Carleton A..................Minneapolis
§ Nelson, Edward N..................Minneapolis
Nelson, Erland R..................Minneapolis
§ Nelson, Harvey..................Minneapolis
§ Nelson, Lloyd S..................Minneapolis
Nelson, Maxine O..................Minneapolis
§ Nelson, Maynard C..................Minneapolis
§ Nelson, O. L. Norman..................Minneapolis
§ Nelson, Wallace I..................Minneapolis
Nerenberg, Sidney..................Minneapolis
§ Nesset, Lawren B..................Minneapolis
§ Nesset, William D..................Minneapolis
§ Neumann, Roland F., Jr..................Minneapolis
§ Neumeister, Charles A..................Minneapolis
§ Nice, Charles M..................Minneapolis
Nolan, Robert K..................Minneapolis
Noran, Axel S. N..................Minneapolis
§ Noran, Harold H..................Minneapolis
§ Nord, Robert E..................Minneapolis
† Nordin, Gustaf T..................Saratoga, Calif.
§ Nordland, Martin..................Minneapolis
§ Nordland, Martin, Jr..................Minneapolis
§ Norman, Mark L., Jr..................Minneapolis
Nuessle, William F..................Minneapolis
§ Nydahl, Malvin J..................Minneapolis
§ Nylander, Emil G..................Minneapolis

§ O'Brien, William A..................Minneapolis
† O'Donnell, James E..................Minneapolis
O'Hanlon, John A..................Minneapolis
§ O'Phelan, E. Harvey..................Minneapolis
*†Oberg, Carl M..................Minneapolis
Olavs, Olga ....Minneapolis
§ Olfelt, Paul C..................Minneapolis
§ Olsen, E. George..................Minneapolis
Olsen, Jay R..................Minneapolis
§ Olson, Alton C..................Minneapolis
§ Olson, Carl J..................Minneapolis
Olson, C. Kent..................Minneapolis
§ Olson, Detlof M..................Minneapolis
† Olson, Olaf A..................Minneapolis
§ Olson, Philip A..................Minneapolis
§ Olson, Rolland A..................Wayzata
§ Oppen, E. Gerhard..................Minneapolis
§ Oppen, Melvin G..................Wayzata
§ Opstad, Earl T..................Unknown
§ Ott, Eugene C..................Minneapolis
Owen, Richard R..................Minneapolis
Paal, Dwain J..................Minneapolis
§ Palen, Benjamin..................Minneapolis
Papermaster, Theodore C..................Minneapolis
Park, Wilford E. ....Minneapolis
§ Pattee, James J..................Minneapolis
§ Peluso, Charles R..................Minneapolis
†§Peppard, Thomas A..................Minneapolis
§ Perlman, Everett C..................Minneapolis
Perlman, Herschel L..................Minneapolis
§ Peteler, Jennings C. L. ....Minneapolis
§ Petersen, Deane A..................Minneapolis
§ Petersen, Glenn L..................Minneapolis
Petersen, Peter C..................Minneapolis
§ Petersen, William E..................Minneapolis
Peterson, Henry ....Minneapolis
Peterson, Herbert W..................Minneapolis
§ Peterson, Nordahl P..................Minneapolis
Peterson, Oliver H..................Minneapolis
Peterson, Oliver H., Jr. ....Minneapolis
§ Peterson, Palmer A..................Minneapolis
†§Peterson, Peter E..................Unknown
§ Peterson, Willard C..................Minneapolis
§ Petit, Julien V..................Minneapolis
†§Petit, Leon J..................Minneapolis
§ Pewters, John T..................Minneapolis
§ Peyton, William T..................Minneapolis
† Phelps, Kenneth A.......Menlo Park, Calif.
Plasha, Matthew K..................Anoka
§ Plass, Herbert F. R..................Minneapolis
†§Platou, Erling S..................Minneapolis
Pleissner, Karl W..................Minneapolis
§ Plimpton, Nathan C., Jr..................Minneapolis
Pohl, John F. M..................Minneapolis
§ Pollak, Kurt ....Minneapolis
§ Pollock, Anthony J..................Minneapolis
† Pollock, David K..................Minneapolis
§ Polzak, Jacob A..................Minneapolis
Popadiuk, Peter..................Minneapolis
† Poppe, Frederick H.......Coral Gables, Fla.
§ Potter, Robert B..................Minneapolis
† Pratt, Fred J..................Minneapolis
§ Pratt, Fred J., Jr..................Minneapolis
† Preine, Irving A..................Osseo
§ Prem, Konald A..................Minneapolis
Preston, Frank S., Jr..................Minneapolis
§ Preston, Paul J..................Minneapolis
§ Price, William E..................Minneapolis
§ Priest, Robert E..................Minneapolis
†§Prim, Joseph A..................Minneapolis
§ Proffitt, William E..................Minneapolis
*§Proshek, Charles E. ....Minneapolis

§ Quello, Robert O..................Minneapolis
§ Quiggle, Arthur B..................Minneapolis
§ Quist, Henry W., Jr..................Minneapolis

Raaen, Olaf..................Minneapolis
Racer, Harley J..................Minneapolis
§ Raile, Richard B..................Minneapolis
§ Ransom, H. Robert..................Osseo
§ Ratelle, Alexander H..................Minneapolis
*§Reader, Donald R..................Minneapolis
‡ Redding, Foster K..................Minneapolis
Reed, Sheldon C..................Minneapolis
Regan, John J..................Minneapolis
§ Regnier, Edward A..................Minneapolis
§ Reif, Harold A..................Minneapolis
§ Reiley, Richard E..................Minneapolis
§ Reiser, Milton P..................St. Louis Park
§ Remole, William D..................Minneapolis
§ Resch, Joseph A. ....Minneapolis
§ Reynolds, Donald P..................Minneapolis
Rholl, Arnold O..................Minneapolis
§ Rice, Carl O..................Minneapolis
Rice, Frank B..................Minneapolis
Rice, Fred A..................Minneapolis
§ Richdorf, Lawrence F..................Minneapolis
§ Rieke, Wellington W..................Wayzata
Riordan, Elsie M..................Minneapolis
§ Risch, Ronald E..................Minneapolis
§ Rizer, Dean K..................Minneapolis
† Rizer, Robert I..................Wayzata
Robb, Edwin F..................Minneapolis
§ Robbins, Owen F..................Minneapolis
§ Roberts, Byron H..................Minneapolis
§ Roberts, Lewis J..................Minneapolis
§ Roberts, Stanley W..................Minneapolis
*†§Roberts, William B..................Minneapolis
Robinson, Cortland O..................Minneapolis
§ Rock, William H..................Anoka
§ Rocknem, Robert E..................Minneapolis
Rockwell, Curtiss V..................Minneapolis
† Rodda, Frederick C. ....Minneapolis
§ Rodgers, Richard S..................Minneapolis
§ Rogin, Norton..................Minneapolis
Rollins, Pat..................Wayzata
§ Romness, Kenneth B..................Mound
§ Rosenbaum, David L..................Minneapolis
§ Rosendahl, Frederick G..................Minneapolis
§ Rosenfield, Abraham B..................Minneapolis

Rosenow, John H.	Minneapolis
† Rosenwald, Reuben M.	Coon Rapids
§ Ross, Alexander J.	Minneapolis
§ Rossen, Ralph X.	Minneapolis
Rossing, Robert G.	St. Paul
Rotenberg, Robert J.	Minneapolis
§ Rothnem, Morris S.	Minneapolis
§ Rucker, William H.	Minneapolis
Rud, Norman E.	Minneapolis
† Rudell, Gustave L.	Minneapolis
§ Russeth, Arthur N.	Minneapolis
§ Rusten, Elmer M.	Minneapolis
Rydburg, Wayne C.	Minneapolis
§ Rydland, Arne D.	Minneapolis
§ St. Cyr, Harry M., Jr.	Robbinsdale
§ St. Cyr, Kenneth J.	Robbinsdale
§ Sadler, William P.	Minneapolis
† Safirescu, Sorin R.	San Francisco, Calif.
§ Saliterman, Bernard I.	Minneapolis
*§Samuelson, Samuel	Minneapolis
§ Sandt, Karl E.	Minneapolis
§ Sawatzky, William A.	Minneapolis
§ Sborov, Abe M.	Minneapolis
§ Scallen, Raymond W.	Minneapolis
§ Schaaf, Frederick H. K.	Minneapolis
Schaar, Frances E.	Minneapolis
Schaefer, Kenneth F.	Minneapolis
Schaefer, Wesley G.	Minneapolis
Schaffhausen, Irwin F.	Minneapolis
Schaffhausen, Mildred	Minneapolis
† Scheldrup, N. H.	Minneapolis
§ Scherer, L. Raymond	Minneapolis
Scherling, Sidney S.	Minneapolis
§ Schiele, Burtrum C.	Minneapolis
§ Schissel, Gregory A.	Minneapolis
§ Schmidt, W. Robert	Minneapolis
† Schmitt, S. C.	Los Angeles, Calif.
Schneck, Jack I.	Minneapolis
§ Schottler, Max E.	Minneapolis
§ Schroeder, Albert J.	Minneapolis
§ Schultz, Alvin L.	Minneapolis
Schultz, Earl A.	Minneapolis
§ Schultz, J. Harold	Minneapolis
§ Schultz, Peter J.	Minneapolis
§ Schulze, William M.	Minneapolis
§ Schumacher, John W.	Minneapolis
§ Schwartz, E. Robert	Minneapolis
§ Schwartz, Virgil J.	Minneapolis
§ Scott, Horace G.	Minneapolis
†§Seaberg, John A.	Pequot Lakes
§ Segal, Martin A.	Minneapolis
Seham, Max	Minneapolis
§ Seifert, Milton H.	Excelsior
§ Selck, Wolfgang T.	Fergus Falls
§ Semsch, Robert D.	Minneapolis
§ Shandorf, James F.	Minneapolis
Shaperman, Eva P.	Minneapolis
§ Shapiro, Irving	Minneapolis
§ Shapiro, Sidney K.	Minneapolis
Sharp, David V.	Minneapolis
§ Shaw, Howard A.	Minneapolis
§ Shea, Andrew W.	Minneapolis
§ Sher, Lewis	Minneapolis
§ Sherman, Lloyd F.	Minneapolis
Sherman, Morris H.	Minneapolis
Shillington, Maurice A.	Brainerd
Shragg, Robert I.	Minneapolis
§ Shronts, John F.	Minneapolis
§ Sidell, Franklin D.	Minneapolis
§ Siegmann, William C.	Minneapolis
§ Silas, Ralph M.	Minneapolis
Silver, John D.	Minneapolis
†§Simons, Jalmar	Unknown
§ Simonson, Donald B.	Minneapolis
† Simpson, Ellery DeW.	Asheville, N. C.
§ Sinykin, Melvin B.	Minneapolis
Siperstein, David M.	Minneapolis
*†Sivertsen, Andrew	Minneapolis
§ Skjold, Arthur C.	Minneapolis
Smiley, John T.	Minneapolis
§ Smisek, Frank M.	Minneapolis
Smith, Adam M.	Minneapolis
§ Smith, Archie M.	Minneapolis
§ Smith, Baxter A., Jr.	Minneapolis
§ Smith, Graham G.	Minneapolis
*†Smith, Homer R.	Minneapolis
† Smith, Margaret I.	Gardena, Calif.
Smith, Nadine G.	Minneapolis
Smith, Norvin R.	Willmar
Smith, Ralph E.	Minneapolis
§ Smith, Theodore S.	Minneapolis
§ Smith, William T.	Minneapolis
§ Soderlind, Ragnar T.	Minneapolis
§ Solhaug, Samuel B.	Minneapolis
Solhaug, Samuel B., Jr.	Minneapolis
§ Solvason, Harold M.	Minneapolis
Spain, W. Thomas	Richmond, Va.
§ Spano, Joseph P.	Minneapolis
§ Spencer, Bernard J.	Minneapolis
§ Spink, Wesley W.	Minneapolis
§ Sponsel, Kenath H.	Minneapolis
† Spratt, Charles N.	Minneapolis
§ Stahr, Aubrey C.	Hopkins
§ Stanford, Charles E.	Minneapolis
† Stanley, Court R.	Minneapolis
Staub, Henry P.	Minneapolis
§ Stelter, Lloyd A.	Minneapolis
§ Stennes, John L.	Minneapolis
† Stenstrom, Annette	Marine-on-the-St. Croix
§ Stephens, William E.	Minneapolis
§ Sterrie, Norman A.	Minneapolis
§ Stewart, Marvin J.	Minneapolis
§ Stewart, Rolla I.	Minneapolis
§ Stiegler, Farrell S.	Minneapolis
§ Stoesser, Albert V.	Minneapolis
§ Stoltz, Robert C.	Minneapolis
† Stomel, Joseph	Los Angeles, Calif.
Stone, Norman F.	Minneapolis
Stone, Stanley P.	Minneapolis
*†Strachauer, Arthur C.	Minneapolis
§ Strickler, Jacob H.	Minneapolis
§ Strom, Gordon W.	Minneapolis
§ Stromgren, Delph T.	Minneapolis
§ Stromme, William B.	Minneapolis
Strunk, Clarence A.	Minneapolis
§ Sturges, Robert L.	Minneapolis
Subak, Barbara H.	Minneapolis
†§Subby, Walter	Wayzata
Sukov, Marvin	Minneapolis
Sullivan, Raymond M.	Minneapolis
Swain, Francis M.	Illinois
Swanson, Roy E.	Minneapolis
Swanson, Wallace L.	Minneapolis
§ Sweetser, Horatio B.	Minneapolis
§ Sweetser, Theodore H.	Minneapolis
§ Sweetser, Theodore H., Jr.	Minneapolis
† Sweitzer, Samuel E.	Minneapolis
† Swendseen, Carl G.	Minneapolis
Syverton, Jerome T.	Minneapolis
Tam, Ernest C.	Minneapolis
§ Tangen, George M.	Minneapolis
Taylor, Joseph H.	Minneapolis
§ Taylor, William E.	Minneapolis
Teeter, Richard R.	Minneapolis
§ Tenner, Robert J.	Minneapolis
§ Tesar, Charles E.	Unknown
Thal, Alan P.	Minneapolis
*†Thomas, George E.	Minneapolis
Thomes, A. Boyd	Minneapolis
Thompson, Arthur	Minneapolis
§ Thompson, Willis H.	Minneapolis
Thorsen, David S.	Minneapolis
§ Thorson, Stuart V.	Minneapolis
§ Thysell, Desmond M.	Minneapolis
§ Tinkham, Robert G.	Minneapolis
§ Titrud, Leonard A.	Minneapolis
Tobin, John D.	Minneapolis
Toon, Robert W.	Minneapolis
Trach, Benedict B.	Minneapolis
§ Trow, James E.	Minneapolis
§ Trow, William H.	Minneapolis
Trueman, Harold S.	Minneapolis
Tsai, Shih Hao	Oak Terrace
§ Tucker, Richard C.	Minneapolis
§ Tudor, Richard B.	Minneapolis
† Turnacliff, Dale D.	St. Paul
§ Twidwell, Joseph E.	Minneapolis
Twomey, John E.	Minneapolis
†§Ulrich, Henry L.	Minneapolis
§ Ulvestad, Harold S.	Minneapolis
†§Undine, Clyde A.	Minneapolis
§ Utendorfer, Robert W.	Minneapolis
Veinbergs, Arnolds	Minneapolis
§ Vennes, Jack A.	Minneapolis
Vik, A. Elliott	Minneapolis
§ Vitols, T. M.	Minneapolis
Wahlquist, Harold F.	Minneapolis
Waldron, Carl W.	Hopkins
§ Wall, Carl R.	Minneapolis
Wallace, Helen M.	Minneapolis
§ Walonick, Albert L.	St Louis Park
§ Walsh, Francis M.	Minneapolis
§ Walsh, William T.	Minneapolis
§ Wangensteen, Owen H.	Minneapolis
Ward, Percy A.	Minneapolis
Waters, Alvin W.	Minneapolis
§ Watson, C. Gordon	Minneapolis
Watson, Cecil J.	Minneapolis
Wattenberg, Lee W.	Minneapolis
Weatherhead, Donald S. P.	Minneapolis
¶ Weaver, Myron Mc.	Schenectady, N. Y.
§ Webb, Edgar A.	Minneapolis
* Webb, Roscoe C.	Minneapolis
§ Webber, Richard J.	St. Louis Park
§ Weber, Lowell W.	Minneapolis
§ Weisberg, Rapheal J.	Minneapolis
§ Wendland, John P.	Minneapolis
Werner, George	Minneapolis
§ Wesolowski, Stanley P.	Minneapolis
† West, Catherine C.	Minneapolis
Westley, Kent	Little Falls
Wetherby, Macnider	Minneapolis
§ Wexler, Harold M.	Minneapolis
§ Wheeler, Robert W.	Minneapolis
Whitacre, John C., II	Minneapolis
§ White, Asher A.	Minneapolis
†§White, S. Marx	Minneapolis
§ White, Willard D.	Minneapolis
§ Whitesell, Lloyd A.	Minneajolis
§ Widen, Wilford F.	Minneapolis
† Wilder, Kenneth W.	Minneapolis
†§Wilder, Robert L.	Minneapolis
† Wiider, R. M., Jr.	Topeka, Kan.
§ Wilder, Walter L.	Minneapolis
§ Wilken, Paul A.	Minneapolis
*†Willcutt, Clarence E.	Phoenix, Ariz.
Williams, Paul A.	Minneapolis
§ Williams, Richard E.	Robbinsdale
Williams, Robert	Unknown
Wilson, Robert E.	Minneapolis
Winchell, Paul	Minneapolis
Winther, Nora M. C.	Minneapolis
§ Wippermann, Frederic F.	Minneapolis
†§Witham, Carl A.	Minneapolis
†§Wittich, Frederick W.	Minneapolis
Wohlrabe, Arthur A.	Minneapolis
§ Wohlrabe, A. Cabot	Minneapolis
§ Wolf, Alfred H.	Minneapolis
Wolter, Frederick H.	Minneapolis
§ Wright, Thomas D.	Minneapolis
§ Wright, Wale S.	Minneapolis
§ Wright, William S.	Edina
* Wyatt, Oswald S.	Minneapolis
Wynne, Herbert M. N.	Minneapolis
§ Ylvisaker, Ragnvald S.	Minneapolis
†§Yoerg, Otto Wm.	Minneapolis
Yue, Wen Y.	Oak Terrace
Zahrendt, O. Lewis	Minneapolis
§ Zarling, V. Richard	Minneapolis
§ Zaworski, Leo A.	Minneapolis
Zierold, Arthur A.	Minneapolis
§ Zinter, Ferdinand A.	Minneapolis
Ziskin, Thomas	Minneapolis

KANDIYOHI-SWIFT-MEEKER COUNTY MEDICAL SOCIETY

Kandiyohi, Swift, and Meeker Counties

Regular meetings, third Thursday. Annual meeting, in December

Number of Members—54

President	
Rorem, Joseph A.	Appleton
Secretary	
Opsahl, Lawrence J.	Willmar
Allison, David D.	Litchfield
Anderson, Richard E.	Willmar
Aulick, Ernest J.	Paynesville
§ Bosland, Howard G.	Willmar
§ Bradley, Nelson J.	Willmar
Carlon, Donald L.	Willmar
Chunn, Stanley S.	Willmar
† Daignault, Oscar	Benson
† Danielson, Karl A.	Litchfield
Danielson, Lennox	Litchfield
Dille, Donald E.	Litchfield
§ Docksey, John W.	Willmar
Eberley, Tobe S.	Benson
§ Ellinger, Albert J.	Willmar
Fedor, Robert D.	Litchfield
§ Ford, F. Wendell	New London
Frederickson, Alice C.	Willmar
§ Gaebe, Milton B.	Clara City
Giere, Silas W.	Benson
§ Gilman, Lloyd C.	Willmar
§ Griffin, Richard P.	Benson
§ Guy, Jack A.	New London
§ Helwig, Karl L., Jr.	Kerkhoven
§ Herbst, Richard F.	Willmar
§ Hinz, Walter E.	Willmar
§ Hodapp, Robert V.	Willmar
§ Holm, Donald F.	Benson
§ Houts, Joseph C.	Dassel

§ Jacobs, Douglas L. .... Willmar
§†Jacobs, Johannes C. .... Willmar
§ Johnson, Marvin W. .... Dassel
Kaufman, Edward J. .... Appleton
§ McCarthy, Austin M. .... Willmar
Macklin, William E., Jr. .... Willmar
§ Meinert, John K. .... Willmar
§ Michels, Roger P. .... Willmar
§ Nelson, Robert H. .... Benson
†§O'Connor, Daniel C. .... Eden Valley
Olson, Gregory M. .... Litchfield
§ Oosahl, Lawrence J. .... Willmar
§ Peterson, Willard E. .... Willmar
† Porter, Oliver M. .... Atwater
§ Proeschel, Ray K. .... Willmar
§ Rorem, Joseph A. .... Appleton
§ Ruchie, Warren H. .... Willmar
Rygh, Harold N. .... Atwater
Schnell, Frederick S. .... Litchfield
†§Solsem, Frederick N. S. .... Spicer
Sutherland, W. H. .... Spicer
Thompson, Russell A. .... Cosmos
Wagner, Norman W. .... Benson
‡ Williams, Francis R. .... Duluth
Wilmot, Cecil A. .... Litchfield
Wilmot, Harold E. .... Litchfield

LYON-LINCOLN COUNTY MEDICAL SOCIETY

Lyon and Lincoln Counties

Regular meetings, 6-week period in spring and fall. Annual meeting, last Tuesday in October

Number of Members—28

President
Larson, Milo H. .... Lake Benton
Secretary
Hedenstrom, Philip C. .... Marshall

§ Bodaski, Albert A. .... Tyler
§ Boyle, Francis J. .... Tracy
§ Eckdale, John E. .... Marshall
§ Ferguson, William C. .... Walnut Grove
§ Ford, Burton C. .... Marshall
† Gray, Frank D. .... Marshall
Guldseth, Gustav J. .... Hendricks
§ Hedenstrom, Philip C. .... Marshall
§ Hermanson, Peter E. .... Hendricks
† Hoidale, Andrew D. .... Tracy
§ Johnson, C. Percy .... Tyler
§ Kreuzer, Titus C. .... Marshall
§ Larson, Milo H. .... Lake Benton
§ Lee,Norman J. .... Tracy
§ Monson, Leonard J. .... Canby
§ Murphy, Joseph E. .... Marshall
§ Myers, John W. .... Canby
Olson, Robert T. .... Canby
§ Peterson, Kenneth A. .... Marshall
§ Remsberg, Robert R. .... Tracy
§ Tainter, Ronald W. .... Marshall
Thill, Leonard J. .... Balaton
§ Thompson, Carl O. .... Hendricks
†§Vadheim, Alfred L. .... Tyler
†§Valentine, Walter H. .... Tracy
§ Wolstan, Simon D. .... Minneota
§ Workman, Warner G. .... Tracy
†§Yaeger, Wilbert W. .... Marshall

McLEOD COUNTY MEDICAL SOCIETY

McLeod County

Regular meetings, third Thursday of each month.
Annual meeting, third Thursday in December

Number of Members—22

President
Howell, Milton M. .... Glencoe
Secretary
Truesdale, Clark W. .... Glencoe

§ Bretzke, Carl O. .... Hutchinson
§ Brink, Donald M. .... Hutchinson
Carroll, John J. .... Winsted
§ Chervenak, William A. .... Winsted
*†Clement, John B. .... Lester Prairie
§ Griebie, Grant L. .... Brownton
§ Howell, Milton M. .... Glencoe
§ Huebert, Dan W. .... Hutchinson
§ Jensen, Alvin M. .... Brownton
*†§Klima, William W. .... Stewart
§ Lippmann, Elmer W. .... Hutchinson
§ McNeil, Maurice A. .... Glencoe
§ Navratil, Donald R. .... Glencoe
§ Neumaier, Arthur A. .... Glencoe
§ Peterson, Kenneth H. .... Hutchinson
Sahr, Walter G. .... Hutchinson
† Scholpp, Otto W. .... Hutchinson
§ Selmo, Joseph D. .... Norwood
§ Sheppard, Charles G. .... Hutchinson
§ Smith, George R. .... Hutchinson
§ Smyth, John J. .... Lester Prairie
§ Truesdale, Clark W. .... Glencoe

MOWER COUNTY MEDICAL SOCIETY

Mower County

Regular Meetings, last Thursday of every month
Annual Meeting, December

Number of Members—38

President
Sheedy, Chester L. .... Austin
Secretary
Rosenthal, F. Harold .... Austin

§ Anderson, David P., Jr. .... Austin
§ Anderson, Harold J. .... Austin
§ Anderson, Wallace R. .... Austin
§ Barber, Tracy E. .... Austin
§ Cronwell, Bernhard J. .... Austin
Elliott, Harold J. .... Hayfield
§ Fetzek, Albert D. .... Austin
§ Fisch, Herbert M. .... Austin
§ Flanagan, Leonard G. .... Austin
§ Grise, William B. .... Austin
§ Hagen, John D. .... Austin
† Havens, John G. W. .... Austin
† Hegge, Olav H. .... Austin
† Hertel, Garfield E. .... Austin
§ Hesla, Inman A. .... Austin
§ Jewell, A. H. .... Austin
§ Leck, Paul C. .... Austin
§ Lommen, Peter A. .... Austin
§ Lommen, Peter A., Jr. .... Austin
§ McKenna, Jay K. .... Austin
† Melzer, George R. .... Lyle
§ Miller, Herman .... Austin
Morse, Morton P. .... LeRoy
§ Nesse, James A. .... Austin
§ Osborn, Donald O. .... Austin
§ Peterson, Stanley C. .... Austin
§ Peterson, W. H. .... Austin
§ Robertson, Paul A. .... Austin
§ Rosenthal, F. Harold .... Austin
§ Sargent, Edward C., Jr. .... Austin
Schneider, Paul J. .... Adams
§ Seery, Thomas M. .... Austin
§ Sheedy, Chester L. .... Austin
§ Stahl, George W. .... Austin
§ Thomson, James M. .... Austin
§ Twiggs, Leo F. .... Austin
§ Van Cleve, Horatio P. .... Austin
§ Wright, Robert R. .... Austin

NICOLLET-LE SUEUR COUNTY MEDICAL SOCIETY

Nicollet and Le Sueur Counties

Regular Meetings, first Monday of alternate months
Annual Meeting, December

Number of Members—30

President
Gridley, John W. .... Arlington
Secretary
Curtis, Rauen A. .... Le Center

† Agar, Ernest A. .... Minneapolis
† Branham, Donald S. .... Deer Park, Wisc.
§ Canfield, Wayne W. .... St. Peter
* Covell, Walter W. .... St. Peter
§ Curtis, Rauen A. .... LeCenter
† Ericson, Swan .... Los Angeles, Calif.
Gislason, Solveg T. .... St. Peter
§ Gridley, John W. .... Arlington
§ Grimes, Burton P. .... St. Peter
Henry, Martin R. .... St. Peter
Hiniker, Peter J. .... Le Sueur
§ Huffington, Herb L., Jr. .... Waterville
* Johnson, Hobart C. .... No. Mankato
† Kabrick, Ola A. .... St. Peter
§ Kath, Reinhard H. .... Arlington
§ Lenander, Mellvin E. .... St. Peter
§ Limbeck, Donald A. .... Le Sueur
Olmanson, Edmund G. .... St. Peter
§ Olmanson, Myron D. .... St. Peter
§ Olson, Duane O. .... Gaylord
§ Rudie, Clifford N. .... St. Peter
Samuelson, L. Gordon .... St. Peter
§ Schulberg, Verne A. .... Gaylord
§ Sjostrom, Lawrence E. .... St. Peter
§ Sonnesyn, Nels N. .... Le Sueur
§ Strathern, Carleton S. .... St. Peter
†§Strathern, Fred P. .... St. Peter
§ Thompson, A. Henry .... St. Peter
Traxler, J. Felix .... Henderson
§ Wohlrabe, Clarence F. .... North Mankato

PARK REGION DISTRICT AND COUNTY MEDICAL SOCIETY

Douglas, Grant, Otter Tail, and Wilkin Counties

Regular meetings, March, June and September. Annual meeting, December
Number of Members—60

President
PARSON, LESTER R. .... Elbow Lake

Secretary
GEISER, PETER M. .... Alexandria

Baker, Jeannette L. .... Fergus Falls
Baker, Norman H. .... Fergus Falls
† Blakey, Adam R. .... Osakis
§ Boline, Clifford A. .... Battle Lake
§ Cain, James H. .... Alexandria
Campbell, Dennis V. .... Fergus Falls
§ Carlson, Carl E. .... Alexandria
Carlson, Homer J. .... Pelican Rapids
Clifford, George W. .... Alexandria
*†§Combacker, Leon C. .... Fergus Falls
§ Daehlin, Rolf .... Fergus Falls
§ DeKruif, Hendrik .... Fergus Falls
§ Doms, Vernon A. .... Elbow Lake
Emerson, Edwin E. .... Osakis
§ Estrem, Ralph L. .... Fergus Falls
§ Estrem, Robert D. .... Fergus Falls
§ Geiser, Peter M. .... Alexandria
§ Hamlon, John S. .... Fergus Falls
Hanson, Everett C. .... New York Mills
† Hanson, LeRoy W. .... Bellaire, Texas
§ Heegaard, William G. .... Alexandria
† Heiberg, Emmett A. .... Fergus Falls
§ Helseth, Hovald K. .... Fergus Falls
§ Hom, Leong, Y. W. .... Battle Lake
§ Hunt, William .... Fergus Falls
§ Jacobson, Clifford W. .... Breckenridge
§ Kevern, Jay L. .... Henning
§ Kippen, Neil .... Breckenridge
§ Korda, Henry A. .... Pelican Rapids
† Larson, Donald M. .... Minneapolis
Leibold, Herbert H. .... Parkers Prairie
§ Lewis, Arthur J. .... Henning
§ Lewis, Charles W. .... Henning
Love, Frederick A. .... Carlos
§ Lund, Carl J. T. .... Fergus Falls
† Mortenson, Nels G. .... Minneapolis
§ Mouritsen, Glenn J. .... Fergus Falls
§ Nelson, Roy A. .... Fergus Falls
Nelson, Wilburn O. B. .... Bisbee, Arizona
§ O'Brien, Louis T. .... Breckenridge
Ostergaard, Erling .... Evansville
Parson, E. Lillian B. .... Elbow Lake
Parson, Lester R. .... Elbow Lake
† Patterson, William L. .... Fergus Falls
† Paulson, Theodore S. .... Fergus Falls
Perkins, Douglass E. .... Alexandria
§ Reinhardt, James H. .... Alexandria
§ Rockwood, Philo H. .... Fergus Falls
§ Sanderson, David J. .... Fergus Falls
† Satersmoen, Theodore .... Pelican Rapids
§ Sather, Edgar R. .... Alexandria
§ Schamber, Walter F. .... Parkers Prairie
Schoeneberger, P. B. .... Perham
§ Sethre, Arthur E. .... Fergus Falls
§ Shaver, Ward .... Fergus Falls
§ Stemsrud, Harold L. .... Alexandria
* Sutton, Harris R. .... Hoffman
§ Tanquist, Edwin J. .... Alexandria
Warner, James J. .... Perham
§ Wasson, Loren F. .... Alexandria

RAMSEY COUNTY MEDICAL SOCIETY

Ramsey County

Regular Meetings, last Monday each month, except June, July, and August
Annual meeting, in January
Number of Members—514

President
LYNCH, FRANCIS W. .... St. Paul

Secretary
SKINNER, ABBOTT .... St. Paul

Exec. Sec'y.
OLSON, MRS. ELEANOR .... St. Paul

§ Abrams, Alexander, Jr. .... St. Paul
§ Adair, Albert F., Jr. .... St. Paul
Adams, Harold R. .... St. Paul
† Ahrens, Albert E. .... St. Paul
§ Ahrens, Robert M. .... St. Paul
§ Alden, John F., Jr. .... St. Paul
Ambrus, Laszlo .... St. Paul
§ Amerongen, W. W. .... St. Paul
§ Arnquist, Andrew S. .... St. Paul
§ Arny, Frederick P. .... St. Paul
§ Arzt, Philip K. .... St. Paul
§ Aurelius, J. Richards .... St. Paul
§ Ausman, Duane R. .... St. Paul
Austrian, Sol .... St. Paul
‡ Axler, Gueri .... New York
§ Babb, Frank S. .... St. Paul
§ Baer, Walter .... St. Paul
Balcome, Milton M. .... St. Paul
Barnett, Joseph M. .... St. Paul
‡ Baronofsky, Ivan D. .... New York
† Barsness, Nellie O. N. .... St. Paul
Bauer, Eugene L. .... St. Paul
†§Beals, Hugh .... LaJolla, Calif.
§ Beck, Charles J. .... North St. Paul
§ Beech, Raymond H. .... St. Paul
§ Beek, Harvey O. .... St. Paul
§ Beer, John J. .... St. Paul
Bell, Charles C. .... St. Paul
§ Bellomo, James .... St. Paul
† Bellomo, John .... St. Paul
§ Benepe, James L. .... St. Paul
Benthack, Elaine M. .... St. Paul
Bernier, M. J. .... North St. Paul
§ Bernstein, William C. .... St. Paul
§ Bicek, Joseph F. .... St. Paul
† Binger, Henry E. .... Phoenix, Ariz.
Bjornson, R. G. B. .... Minneapolis
Black, Earl J. .... St. Paul
Blackburn, Henry W. .... St. Paul
Blomberg, William R. .... St. Paul
Blumberg, Henry B. .... St. Paul
† Bock, Rolland A. .... St. Paul
*§Bolender, Harold L. .... St. Paul
§ Bonello, Frank J. .... St. Paul
§ Borg, Joseph F. .... St. Paul
† Bouma, Lewis R. .... St. Paul
Bouthilet, Florence J. .... St. Paul
Brand, George D. .... St. Paul
† Bray, Elwyn R. .... St. Paul
§ Briggs, John F. .... St. Paul
§ Broadie, Thomas E. .... St. Paul
§ Brodie, Walter D. .... St. Paul
Brown, Ian A. .... St. Paul
‡ Brown, James E., Jr. .... St. Paul
† Brown, John C. .... Los Gatos, Calif.
Buckley, J. J. .... Edina
§ Bulinski, Theodore J. .... St. Paul
§ Burch, Edward P., II .... St. Paul
*†Burch, Frank E. .... St. Paul
§ Burkland, Edwin C. .... St. Paul
§ Burlingame, David A. .... St. Paul
§ Burneister, Richard O. .... St. Paul
† Burns, Robert M. .... St. Paul
Burton, Carl G. .... St. Paul
§ Bush, Robert P. .... St. Paul
§ Busher, Herbert H. .... St. Paul
§ Cain, Clark L. .... St. Paul
Calin, Stanford H. .... St. Paul
Callahan, Francis F. .... St. Paul
Cameron, Dale C. .... St. Paul
Canine, James L. .... So. St. Paul
§ Carley, Walter A. .... St. Paul
Cederleaf, Cherry B. .... Mahtomedi
Chadbourn, Charles R. .... St. Paul
†§Chatterton, Carl C. .... St. Paul
§ Christiansen, Andrew .... St. Paul
§ Clark, Henry B., Jr. .... Minneapolis
§ Cochrane, Byron B. .... St. Paul
Coddon, Walter D. .... St. Paul
§ Cohen, Ellis N. .... St. Paul
§ Colby, Woodard L. .... St. Paul
Cole, Wallace H. .... St. Paul
§ Coleman, John B. .... St. Paul
† Collie, Henry G. .... St. Petersburg, Fla.
§ Connolly, Coleman J. .... St. Paul
Connolly, Joseph P. .... So. St. Paul
Connor, Charles E. .... St. Paul
§ Cook, C. Kenneth .... St. Paul
§ Cooper, Charles C. .... St. Paul
‡ Coseriu, Vasile G. .... St. Paul
§ Craig, David M. .... St. Paul
Crowley, James H. .... St. Paul
§ Crudo, Vincent D. .... St. Paul
§ Crump, James W. .... St. Paul
*§Culligan, John M. .... St. Paul
§ Culver, L. G. .... St. Paul
§ Cumming, E. Dale .... St. Paul
§ Davis, Edward V. .... St. Paul
Dawson, James R. .... St. Paul
Decker, Charles H. .... St. Paul
§ Derauf, Benjamin I. .... St. Paul
§ Derauf, Donald E. .... St. Paul
§ Deters, Donald C. .... St. Paul
§ Drake, Carl B. .... St. Paul
*§Dunn, James N. .... St. Paul
Dunn, Robert C. .... St. Paul
Dyrdal, Paul J. .... St. Paul
§ Earl, George A. .... St. Paul
§ Earl, John R. .... St. Paul
Edwards, Joseph W. .... St. Paul
Edwards, Lloyd G. .... St. Paul
§ Edwards, Thomas J. .... St. Paul
§ Eginton, Charles T. .... St. Paul
* Ely, Orriman S. .... So. St. Paul
Emerson, Edward C. .... St. Paul
§ Emmons, R. W. .... St. Paul
§ Endress, Edward K. .... St. Paul
§ Enroth, Oscar E. .... St. Paul
† Ernest, George C. H. ....
St. Petersburg, Fla.
§ Ersfeld, Murray P. .... St. Paul
† Eshelby, E. C. .... St. Paul
Farkas, John V. .... St. Paul
§ Fee, John G. .... St. Paul
§ Feinberg, Milton .... Sacramento, Calif.
§ Felder, Davitt A. .... St. Paul
§ Felion, Arthur J. .... St. Paul
§ Fesler, Harold H. .... St. Paul
Field, Anthony H. .... Farmington
§ Fink, Daniel L. .... St. Paul
§ Fischer, Robert F. .... St. Paul
§ Fisher, Dan W. .... St. Paul
§ Flanagan, Harold F. .... St. Paul
§ Flannery, Hubert F. .... St. Paul
§ Flom, Reynold P. .... St. Paul
§ Flynn, L. L., Jr. .... St. Paul
† Fogarty, Charles W. .... St. Paul
§ Fogarty, Charles W., Jr. .... St. Paul
§ Fogelberg, Emil J. .... St. Paul
§ Foley, Frederic E. B. .... St. Pau
Forsythe, James R. .... St. Pa
§ Fox, LeRoy J. .... St. Pau
§ Freeman, Charles D., Jr. .... St. Pau
§ Freeman, Gerald I. .... St. Paul
§ Freidman, Louis L. .... St. Paul
§ Fritz, Wallace L. .... St. Paul
§ Froats, Charles W. .... St. Paul
§ Fuller, Benjamin F. .... St. Paul
§ Galligan, John J. .... St. Paul
§ Garbrecht, Arthur W. .... St. Paul
§ Gardner, Walter P. .... St. Paul
Garrow, Douglas M. .... St. Paul
§ Gehlen, Joseph N. .... St. Paul
Gibbs, Edward C. .... St. Paul
§ Gillespie, Delmar R. .... St. Paul
Gilsdorf, Donald A. .... St. Paul
§ Gleason, Wallace A. .... St. Paul
§ Goldsmith, Joseph W. .... St. Paul
†§Goltz, Edward V. .... St. Paul
§ Grant, Hendrie W. .... St. Paul
†§Gratzek, Thomas .... St. Paul
§ Grau, Robert K. .... St. Paul
Gray, Edward F. .... White Bear Lake
Hagen, Paul S. .... St. Paul
§ Hakanson, Erick Y. .... St. Paul
§ Hall, Barnard .... St. Paul
§ Hammes, Ernest M. .... St. Paul
§ Hammes, Ernest M., Jr. .... St. Paul
§ Hannon, Donald W. .... St. Paul
Harbaugh, John T. .... Neosho, Mo.
§ Harmon, Gaius E. .... St. Paul
§ Hartfiel, William F. .... St. Paul
Hartig, Marjorie .... St. Paul
§ Hauser, Victor P. .... St. Paul
§ Hayes, Albert F. .... St. Paul
§ Heck, William W. .... St. Paul
Hedenstrom, Frank G. .... St. Paul
Heilig, William R. .... St. Paul
Henderson, Arthur J. G. .... North St. Paul

§ Hengstler, William H. ....St. Paul
† Hensel, Charles N. ....St. Paul
Herman, Samuel M. ....St. Paul
Herrmann, Edgar T. ....St. Paul
Hertz, Myron J. ....St. Paul
*†Hilger, Andrew W. ....St. Paul
§ Hilger, Jerome A. ....St. Paul
Hilger, Laurence D. ....St. Paul
§ Hilker, Marcus D. ....St. Paul
Hiniker, Louis P. ....St. Paul
§ Hochfilzer, John J. ....St. Paul
Hodgson, Jane E. ....St. Paul
† Holcomb, O. William ....St. Paul
Hollinshead, W. H. ....St. Paul
§ Holmen, Robert W. ....St. Paul
§ Holt, John E. ....St. Paul
Hopkins, G. Wendell ....St. Paul
Howard, Merrill A. ....St. Paul
§ Howe, Newell W. ....St. Paul
† Hoyt, Charles S. ....Rochester
§ Hullsiek, Harold E. ....St. Paul
*§Hullsiek, Richard B. ....Fort Snelling
Hunter, Murray H. ....Farmington
§ Hunter, Samuel W. ....St. Paul
§ Hurwitz, Milton M. ....St. Paul
§ Husebye, Kjeld O. ....St. Paul
† Ide, Arthur W. ....White Bear Lake
Ikeda, Kano ....St. Paul

Jackson, William C. ....St. Paul
§ Jahnke, William R. ....St. Paul
§ James, Ellery M. ....St. Paul
§ James, John W. ....St. Paul
§ Janssen, Martin E. ....St. Paul
†§Jarvis, Bruce W. ....St. Paul
Jarvis, Charles W. ....St. Paul
Jarvis, Marilyn A. ....St. Paul
§ Jastram, Rupert M. ....St. Paul
† Jesion, Joseph W. ....Pine River
§ Johanson, Waldemar G. ....St. Paul
Johnson, Carl E. ....St. Paul
Johnson, Carolyn A. ....St. Paul
§ Johnson, Herbert W. ....St. Paul
§ Johnson, R. J. ....St. Paul
† Jones, E. Mendelssohn ....St. Paul

§ Kamman, Gordon R. ....St. Paul
Kaplan, David H. ....St. Paul
Karon, Irvine M. ....St. Paul
§ Kasper, Eugene M. ....St. Paul
Katz, Louis J. ....Long Beach, Calif.
Keefe, Rolland E. ....St. Paul
§ Kelly, Albert C. ....St. Paul
§ Kelly, Edward H. ....St. Paul
Kelly, James H. ....St. Paul
*†§Kelly, John V. ....St. Paul
§ Kelsey, Chauncey M. ....St. Paul
§ Kenefick, Emmett V. ....St. Paul
§ Kenyon, Thomas J. ....St. Paul
§ Kesting, Herman ....St. Paul
† King, George L. ....Hudson, Wisc.
§ Knutson, Gerhard E. ....St. Paul
§ Knutson, Robert C. ....St. Paul
Kodres, Nina ....St. Paul
Koza, Donald W. ....St. Paul
§ Krezowski, Thomas K. ....St. Paul
§ Kugler, Alex A. ....St. Paul
§ Kuske, Albert W. ....St. Paul
§ Kusske, Bradley W. ....St. Paul
§ Kusske, Douglas R. ....St. Paul
* Kvitrud, Gilbert ....St. Paul

§ Lannin, Bernard G. ....St. Paul
§ Lannin, Donald R. ....St. Paul
§ Larrabee, Walter F., Jr. ....St. Paul
Larson, Eva-Jane Ostergren ....St. Paul
Larson, James T. ....So. St. Paul
Larson, Kenneth R. ....St. Paul
Larson, Martin L. ....St. Paul
Laszewski, Franz von Zelberschwecht St. Paul
†§Leahy, Bartholomew ....St. Paul
*§Leavenworth, Richard O. ....St. Paul
§ Leick, Richard M. ....St. Paul
Leitch, Archibald ....St. Paul
§ Lepak, John A. ....St. Paul
† Lerche, William..................Cable, Wisc.
Leven, N. Logan ....St. Paul
§ Leverenz, Carleton W. ....St. Paul
Levitt, George X. ....St. Paul
§ Lick, Charles L. ....St. Paul
§ Lick, Louis C. ....St. Paul
§ Lick, William J., Jr. ....St. Paul
§ Lien, Richard J. ....St. Paul
† Lightbourn, Edgar L. ....Hastings
Lilleberg, Norbert J. ....St. Paul
§ Lindell, Robert ....St. Paul
Lippman, Hyman S. ....St. Paul
Litkewitsch, Helene ....St. Paul
§ Loken, Selmer M. ....St. Paul
Lowe, Earl R. ....So. St. Paul
Lowe, Thomas A. ....So. St. Paul
§ Lundholm, Arthur M. ....Harris
§ Lynch, Francis W. ....St. Paul

§ McCabe, James S. ....St. Paul
§ McCain, Donovan L. ....St. Paul
McCarthy, Joseph J. ....St. Paul
† McClanahan, James H...White Bear Lake
McClanahan, Thomas S...White Bear Lake
McClellan, Robert J. ....St. Paul
McCloud, Charles N., Jr. ....St. Paul
McDaniel, S. P. ....Lakeville
§ McEwan, Alexander ....St. Paul
§ McGroarty, Brian J. ....St. Paul
McKenzie, Eva E. ....St. Paul
§ McNeill, J. A. ....St. Paul
MacLean, Lloyd D. ....St. Paul
Mackoff, Sam M. ....Phoenix, Arizona
Madland, Robert S. ....St. Paul
§ Malerich, J. Anthony ....St. Paul
§ Malerich, J. Anthony, Jr. ....St. Paul
§ Manlove, Charles H., Jr. ....St. Paul
Mark, Hilbert ....Toledo, Ohio
Marks, Roger W. ....St. Paul
§ Martin, Dwight L. ....St. Paul
†§Martineau, Joseph L. ....St. Paul
§ Mateo, Guillermo ....St. Paul
Matthews, James H. ....Minneapolis
§ Mazzitello, William F. ....St. Paul
*§Meade, John R. ....St. Paul
§ Mears, Burtis J. ....St. Paul
§ Medelman, John P. ....St. Paul
§ Melancon, Joseph F. ....St. Paul
§ Menold, William F. ....St. Paul
§ Merner, Thomas B. ....Faribault
§ Merrick, Robert L. ....St. Paul
§†Meyerding, Edward A. ....St. Paul
§ Michienzi, Leonard J. ....St. Paul
Midboe, Gilbert ....St. Paul
§ Miller, Albert G. ....St. Paul
Miller, William T. ....St. Paul
Miller, Z. R. ....St. Paul
§ Milnar, Frank J. ....St. Paul
§ Mishek, Charles J. ....St. Paul
§ Moga, John A. ....St. Paul
Molander, Herbert A. ....St. Paul
§ Monahan, Robert H. ....St. Paul
§ Mooney, Robert D. ....St. Paul
† Moquin, Marie A. ....St. Paul
† Moren, J. Adelaide ....White Bear Lake
Moriarty, Berenice ....St. Paul
Moriarty, Cecile R. ....St. Paul
§ Muller, A. Eugene ....North St. Paul
Mundahl, Harold R. ....St. Paul
§ Murphy, Jack T. ....St. Paul
§ Nash, Leo A. ....St. Paul
Neibergs, Lidija ....St. Paul
Neibergs, Pauls ....St. Paul
§ Nelson, Loren E. ....St. Paul
† Nelson, Louis A. ....St. Paul
§ Nelson, Louis A., Jr. ....St. Paul
Nimlos, Kenneth O. ....St. Paul
† Nimlos, Lenore O. ....St. Paul
§ Noble, John F. ....St. Paul
§ Noble, J. Lawrence ....St. Paul
§ Norman, David D. ....St. Paul
Nuebel, Charles J. ....St. Paul
Nye, Katherine A. ....St. Paul
Nye, Lillian L. ....St. Paul
§ O'Brien, J. C. ....St. Paul
*†O'Connor, Loren J. ....St. Paul
O'Kane, Thomas W. ....St. Paul
§ O'Malley, Valentine ....St. Paul
§ O'Reilley, Bernard E. ....St. Paul
§ Ockuly, Orville ....St. Paul
†§Ogden, Warner ....St. Paul
†§Ohage, Justus ....St. Paul
§ Olsen, Ralph L. ....St. Paul
Olson, Charles A. ....St. Paul
§ Ostergren, Edward W. ....St. Paul
§ Ouellette, Alfred J. ....St. Paul
§ Owens, Frederick M., Jr. ....St. Paul

§ Palm, Neil M. ....St. Paul
Paulson, Elmer C. ....St. Paul
Paulson, Wallace J. ....Minneapolis
Pearson, Fritz R. ....St. Paul
§ Pearson, Malcolm M. ....St. Paul
§ Peck, Willard R. ....St. Paul
Pedersen, Arthur H. ....St. Paul
Peterson, David B. ....St. Paul
§ Peterson, Donald H. ....St. Paul
§ Peterson, Edward A. ....St. Paul
§ Peterson, Harold O. ....St. Paul
Peterson, Joel L. E. ....St. Paul
Peterson, Roy L. ....White Bear Lake
§ Plotke, Harry L. ....St. Paul
Polski, Paul G. ....So. St. Paul
§ Post, Edmund A. ....St. Paul
§ Proud, Harry S. ....St. Paul

Quattlebaum, Frank W. ....St. Paul

§ Ralph, James R. ....St. Paul
Ramlow, Ralph M. ....St. Paul
† Ramsey, Walter R. ....St. Paul
Rasmussen, Ramby C. ....St. Paul
§ Ravits, Harold G. ....St. Paul
§ Rea, Charles E. ....St. Paul
§ Reid, James W. ....So. St. Paul
§ Reif, Robert W. ....White Bear Lake
Richards, Albert M. ....St. Paul
Richards, Ernest T. F. ....St. Paul
Richardson, Edward J., Jr. ....St. Paul
§ Richardson, Robert J. ....St. Paul
Rick, Paul F. W. ....St. Paul
Rinkey, Eugene ....St. Paul
Ripple, Rudolph J. ....St. Paul
§ Ritchie, Wallace P. ....St. Paul
§ Ritt, Albert E. ....St. Paul
§ Roach, Donald E. ....St. Paul
Robbins, Mason C. ....St. Paul
*§Rogers, Sydney F. ....St. Paul
Rolig, David H. ....St. Paul
§ Rollie, Orris O. ....St. Paul
§ Rosenthal, Robert ....St. Paul
§ Roth, George C. ....St. Paul
†§Rothschild, Harold J. ....St. Paul
§ Rowe, Clarence J., Jr. ....St. Paul
§ Roy, Phil C. ....St. Paul
† Ruhberg, George N. .... Santa Barbara, Calif.
§ Rukavina, John G. ....St. Paul
§ Rusterholz, Alan P. ....St. Paul
§ Ryan, John J. ....St. Paul
§ Ryan, Joseph M. ....St. Paul
§ Sarnecki, M. M. ....St. Paul
† Satterlund, Victor L. .... New Richmond, Wisc.
†§Savage, Francis J. ....St. Paul
§ Schmidtke, Reinhardt L. ....St. Paul
† Schoch, Robert B. J. ....St. Paul
§ Schons, Edward ....St. Paul
§ Schroeckenstein, Hugo F. ....St. Paul
†§Schuldt, Frederick C. ....St. Paul
† Schulze, Albert G. ....St. Paul
§ Schwyzer, Hanns C. ....St. Paul
Schwyzer, Marguerite ....St. Paul
§ Scott, Eugene E. ....St. Paul
Sekhon, Mohan S. ....St. Paul
§ Sells, Richard J. ....North St. Paul
§ Setzer, Hobert J. ....St. Paul
Shannon, William R. ....St. Paul
§ Shelander, Marcus I. ....St. Paul
† Shellman, John J. .... Pacific Palisades, Calif.
Short, Jacob ....St. Paul
Siegel, Clarence ....St. Paul
§ Simons, Leander T. ....St. Paul
Singer, Benjamin J. ....St. Paul
§ Skinner, Abbott ....St. Paul
† Skinner, Harvey O. ....St. Paul
† Skworcow, George ....St. Paul
§ Smisek, Elmer A. ....St. Paul
§ Smith, Vernon D. E. ....St. Paul
§ Snyder, George William ....St. Paul
§ Sohlberg, Olof I. ....St. Paul
§ Sommerdorf, Vernon L. ....St. Paul
§ Sommers, Ben ....St. Paul
§ Sorem, Milton B. ....St. Paul
Soucheray, Philip H. ....St. Paul
§ Souster, Benjamin B. ....St. Paul
§ Sowada, Ernest J. ....St. Paul
Sprafka, Gregory ....St. Paul
§ Sprafka, Joseph L. ....St. Paul
* Sprafka, Joseph M. ....St. Paul
§ Steinberg, Charles L. ....St. Paul
§ Sterner, Donald C. ....St. Paul
§ Sterner, E. R. ....St. Paul
§ Sterner, John J. ....St. Paul
Stewart, Alexander ....St. Paul
§ Stolpestad, Armer H. ....St. Paul
*§Stolpestad, Herbert L. ....St. Paul
§ Strand, Jack W. ....St. Paul
§ Strate, Gordon E. ....St. Paul
Straus, M. L. ....St. Paul
§ Strem, Edward L. ....St. Paul
§ Sturley, Rodney F. ....St. Paul
Sullivan, William A., Jr. ....Minneapolis
† Swanson, John A. ....St. Paul
§ Swanson, Lawrence J. ....St. Paul
Swendson, James J. ....St. Paul
Swenson, Donald B. ....St. Paul
Tani, George ....St. Paul
§ Teisberg, John E. ....St. Paul
Thompson, Floyd A. ....St. Paul
Thoreson, M. C. Bernice ....So. St. Paul
Tiffany, Francis B. ....St. Paul
§ Tifft, Cyril R. ....St. Paul
§ Tongen, Lyle A. ....St. Paul
Travis, James S. ....St. Paul
§ Tregilgas, Harold R. ....So. St. Paul
Tregilgas, Richard B. ....St. Paul

§ Ubel, Frank A. ....St. Paul

Van Bergen, Frederick H. ....Minneapolis
Varco, Richard L. ....St. Paul
§ Vaughn, C. Gordon ....St. Paul
Veirs, Dean M. ....St. Paul
Veirs, Ruby J. S. ....St. Paul
§ Venables, Alexander E. ....St. Paul
Votel, Thomas W. ....St. Paul

§ Waas, Charles W. ....St. Paul
§ Walker, Arthur E. ....St. Paul
§ Wall, James O. ....St. Paul
§ Wallinga, Jack V. ....St. Paul
§ Walsh, Edward F. ....St. Paul
§ Walter, Clarence W. ....St. Paul
Warren, Cecil A. ....St. Paul

Watson, P. Theodore	St. Paul
§ Watson, William H. A.	St. Paul
§ Watson, William J.	Newport
Watz, Clarence E.	St. Paul
§ Webber, Fred L.	St. Paul
§ Wedes, Deno J.	St. Paul
§ Weis, Benjamin A.	St. Paul
§ Weisberg, Maurice	St. Paul
§ Wenzel, Gilbert P.	St. Paul
§ Westover, D. E.	St. Paul
Wetteland, Thomas F.	W. St. Paul
§ Wetzel, Earl V.	St. Paul
† Williams, Arthur B.	St. Paul
§ Williams, Clayton K.	St. Paul
§ Williams, George E.	St. Paul
§ Williams, John A.	St. Paul
Williams, Richard A.	St. Paul Park
§ Wilson, J. Allen	St. Paul
§ Wilson, James V.	St. Paul
† Winnick, Joseph B.	St. Paul
§ Witthaus, Melvyn E.	St. Paul
Wolkoff, H. J.	St. Paul
Word, Harlan L.	St. Paul
Youngren, Everett R.	St. Paul
Zachman, Leo L.	St. Paul
Zagaria, James F.	St. Paul
§ Zimmermann, Bernard D.	St. Paul
†§Zimmermann, Harry B.	St. Paul

RED RIVER VALLEY MEDICAL SOCIETY

Kittson, Mahnomen, Marshall, Norman, Pennington, Polk, Red Lake and Roseau Counties

Regular meetings, quarterly. Annual meeting, December
Number of Members—60

President	
Kinkade, Byron R.	Ada
Secretary	
Sather, Russell O.	Crookston
§ Anderson, Wallace E.	Clearbrook
§ Behr, Orlo K.	Crookston
§ Berg, Arnold M.	Roseau
§ Berge, David O.	Roseau
† Biedermann, Jacob	Thief River Falls
Boroson, Hugh N.	Thief River Falls
§ Boyer, George S.	Crookston
† Bratrud, Edward	Thief River Falls
Brink, Adlai A.	Baudette
§ Cameron, John H.	Crookston
§ Clapp, Hubert D.	Crookston
§ Dale, Lester N.	Red Lake Falls
*†Delmore, John L.	Inglewood, Calif.
§ Delmore, John L., Jr.	Roseau
† Delmore, Robert J.	New Orleans, La.
† Efteland, Myles	New Haven, Conn.
§ Erickson, Eskil	Halstad
§ Feigal, William M.	Thief River Falls
Flancher, Leon H.	Crookston
Grasse, John M., Jr.	Aibonito, Puerto Rico
Giesbrecht, A.	Hallock
§ Greene, Daniel E.	Thief River Falls
§ Hirsh, Stanton A.	Crookston
§ Holmstrom, Carle H.	Warren
§ Jancky, Allen G.	Baudette
§ Jensen, John A.	Crookston
§ Kinkade, Byron R.	Ada
§ Klefstad, Lloyd H.	Greenbush
§ Kostick, William R.	Fertile
§ Loken, Theodore	Ada
§ McKaig, Alan M.	Red Lake Falls
§ Martin, George B.	Thief River Falls
§ Mercil, William F.	Crookston
Mochal, Milo A.	Thief River Falls
§ Mueller, Donald R.	Bagley
† Nelson, Henry E.	Crookston
§ Nelson, Kenneth L.	College Station, Texas
§ Norquist, Joseph L.	Thief River Falls
§ Oppegaard, Chester L.	Crookston
Potts, L. C.	Warroad
§ Pumala, Erven E.	Warren
§ Reff, Alan R.	Crookston
§ Roholt, Christian	McIntosh
§ Roholt, H. B.	Fosston
Sather, Edgar L.	Fosston
§ Sather, George A.	Fosston
§ Sather, Richard N.	Fosston
§ Sather, Russell O.	Crookston
Scheuneman, Allen F.	Warroad
§ Schnabel, Robert F.	Crookston
§ Schossow, George W.	Erskine
§ Skogerboe, Rudolph B.	Karlstad
§ Stadem, Clifford J.	Twin Valley
§ Starekow, Milton D.	Thief River Falls
§ Stensgaard, Kermit L.	Thief River Falls
§ Stewart, Donald E.	Crookston
§ Thysell, Harold R.	Crookston
§ Uhley, Charles G.	Crookston
§ Van Rooy, George T.	Thief River Falls
§ Wikoff, Howard M.	Crookston

RENVILLE-REDWOOD COUNTY MEDICAL SOCIETY

Renville and Redwood Counties

Regular meetings, third Tuesday of each month. Annual meeting, December

Number of Members—26

President	
Metz, Donald D.	Buffalo Lake
Secretary	
Alton, Donald G.	Bird Island
§ Alcorn, William J.	Wabasso
§ Alton, Donald G.	Bird Island
§ Anderson, Chester A.	Hector
§ Billings, Ralph E.	Franklin
† Brand, William A.	Redwood Falls
§ Ceplecha, Stanley F.	Redwood Falls
§ Cosgriff, James A.	Olivia
Cosgriff, James A., Jr.	Olivia
§ Diessner, Ardell W.	Redwood Falls
§ Dordal, John	Sacred Heart
§ Fawcett, Arthur M.	Renville
§ Flinn, James B.	Redwood Falls
† Flinn, Thomas E.	Remer
† Furr, Leo R.	Unknown
§ Haas, Jack F.	Northfield
§ Hinderaker, Harris P.	Bird Island
§ Inglis, William	Redwood Falls
Johnson, Orville H.	Edina
§ Johnson, William E.	Morgan
§ Knoche, Harvey A.	Morgan
§ Lenz, Joseph R.	Morton
§ Metz, Donald D.	Buffalo Lake
§ Nelson, Glenn E.	Fairfax
§ Pierce, Robert B.	Renville
† Preisinger, Joseph W.	Dallas, Texas
§ Strauchler, Jonas	Belview

RICE COUNTY MEDICAL SOCIETY

Rice County

Regular meetings, third Tuesday of each month. Annual meeting, third Tuesday in October

Number of Members—38

President	
Beaton, J. Gordon	Northfield
Secretary	
Smith, Thorsten	Faribault
§ Bauer, Paul G.	Faribault
§ Beaton, J. Gordon	Northfield
§ Bruhl, Heinz H.	Faribault
Buesgens, Ralph H.	Waterville
†§Dungay, Neil S.	Northfield
§ Engberg, Edward J.	Faribault
Francis, David W.	Morristown
§ Furlow, William L.	Faribault
†§Hanson, Adolph M.	Faribault
§ Hanson, John W.	Northfield
† Huxley, Frederick R.	Faribault
Kennedy, George L.	Faribault
§ Kolars, James J.	Faribault
‡ Kucera, Louis B.	Colorado Springs, Colo.
§ Larson, Wyllis G.	Northfield
§ Lende, Norman	Faribault
*†Lexa, F. J.	Lonsdale
*§Mears, Robert F.	Northfield
§ Meyer, Frederick C.	Kenyon
Meyer, Paul F.	Faribault
Meyer, Robert P.	Faribault
§ Mosses, Royal R.	Kenyon
§ Nelson, Ernest J.	Lonsdale
§ Nielsen, Alvin M.	Northfield
§ Nuetzman, Arthur W.	Faribault
§ Orr, Burton A.	Faribault
Ozolins, Marta	Faribault
Pasek, Edward A.	Faribault
Petersen, Donald H.	Northfield
§ Robilliard, Charles M.	Faribault
Rumpf, Carl W.	Faribault
Rysgaard, George N.	Northfield
§ Smith, Thorsten	Faribault
§ Street, Bernard	Northfield
Studer, Donald J.	Faribault
§ Traeger, Carl A.	Faribault
Weaver, Paul H.	Faribault
§ Wilson, Warren E.	Northfield

ST. LOUIS COUNTY MEDICAL SOCIETY

Carlton, Cook, Itasca, Lake and St. Louis Counties

Regular meetings, second Thursday except July and August.
Annual meeting, second Thursday in January

Number of Members—305

President	
Feuling, John C.	Duluth
Secretary	
Bergan, Robert O.	Duluth
Exec. Sec'y	
Gilbert, Mrs. Margaret B.	Duluth
§ Abraham, Arden L.	Duluth
†§Adams, Bertram S.	Hibbing
§ Addy, Edward R.	Gilbert
§ Ahola, Kenneth E.	Hibbing
§ Ahrens, Curtis F.	Duluth
§ Alpert, Abraham H.	Virginia
§ Anderson, Richard W.	Aurora
§ Andrew, William F.	Duluth
§ Antonow, Arthur M.	Virginia
§ Asta, Joseph J.	Duluth
§ Athens, Alvin G.	Duluth
§ Atmore, William G.	Duluth
§ Aufderheide, Arthur C.	Duluth
§ Backus, Reno W.	Nopeming

§ Bagley, Charles M.	Duluth
Bagley, Elizabeth C.	Duluth
§ Bagley, William R.	Duluth
Baich, Velemir M.	Coleraine
§ Bakkila, Henry E.	Duluth
§ Balmer, Albert I.	Duluth
§ Bardon, Richard	Duluth
§ Barker, John D.	Duluth
§ Barnes, Richard E.	Aurora
† Barney, Leon A.	Duluth
§ Barrett, Earl E.	Duluth
§ Bartzen, Peter J.	Duluth
§ Becker, Frederick T.	Duluth
§ Benell, Otto E.	Virginia
Bepko, Marie K.	Cloquet
Berdez, George L.	Duluth
§ Bergan, Robert O.	Duluth
§ Bianco, Anthony J.	Duluth
† Binet, Henry E.	Grand Rapids
§ Blackmore, Sidney C.	Biwabik
§ Bloom, Joseph	Silver Bay
Bolz, J. Arnold	Grand Rapids
Boman, Paul G.	Duluth
§ Bonner, John L.	Eveleth
§ Bouchelle, McLemore	Virginia
Bowen, Robert L.	Hibbing
§ Boyer, Sam H., Jr.	Duluth
§ Braun, Ohrmundt C.	Grand Rapids
§ Bray, Philip N.	Duluth
§ Brockway, Roger W.	Grand Rapids
† Brooker, Warren J.	Minneapolis
§ Brown, Cyrus C., Jr.	Duluth
§ Buckley, Robert P.	Duluth
† Burleigh, Edward G.	Minneapolis
§ Butler, John K.	Cloquet
Callan, Joseph A.	Virginia
Cantwell, William F.	International Falls
§ Chermak, Francis G.	International Falls
§ Chittum, John R.	Aurora
§ Christensen, Clarence H.	Duluth
Ciriacy, Edward W.	Ely
§ Clark, Clarence L.	Duluth
Clark, Elizabeth A.	Duluth
§ Clark, Ivan T.	Duluth
§ Coll, James J.	Duluth
† Collins, Arthur N.	Duluth
§ Conley, Francis W.	Duluth
§ Cope, Hershel B.	Virginia
Coventry, William D.	Duluth
§ Cowan, George M.	Duluth
§ Detjen, Edward D.	Bigfork
§ Deutsch, Robert J.	Duluth
§ Dickson, Franklin H., Jr.	Proctor
Dobler, Manfred G.	Ely
† Doxsee, George C.	Iowa City, Iowa
§ Dwyer, John J.	Duluth
§ Eckman, Philip F.	Duluth
§ Eckman, Ralph J.	Duluth
§ Eisenman, Walter	Hibbing
† Ekblad, John W.	Rock Island, Ill.
§ Eklund, Carl D.	Duluth
§ Elias, Frank J.	Duluth
§ Emanuel, Karl W.	Duluth
Eppard, Raymond M.	Cloquet
§ Erickson, George P.	Hibbing
§ Erickson, Vernon D.	Grand Rapids
†§Erskine, Gordon M.	Grand Rapids
§ Evansta, John B.	Grand Rapids
Ewens, George B.	Virginia
§ Fawcett, Keith	Duluth
§ Fellows, Manley F.	Duluth
Ferrand, Paula T.	Moose Lake
Ferrell, Clarence R.	Grand Rapids
§ Feuling, John C.	Duluth
§ Fifield, Malcolm M.	Duluth
§ Fischer, Mario McC.	Duluth
§ Fisketti, Henry	Duluth
§ Flynn, Bernard F.	Hibbing
§ French, Bayard T.	Hibbing
§ Fuller, Josiah	Duluth
§ Gillespie, Malcolm G.	Duluth
§ Goldish, Daniel R.	Duluth
§ Goldish, Robert J.	Duluth
§ Goldschmidt, Volker	Duluth
§ Goodman, Charles E.	Virginia
§ Goodnow, William H.	Duluth
§ Gowan, Lawrence R.	Duluth
†§Graham, Archibald W.	Chisholm
§ Grahek, Jack P.	Ely
†§Granquist, Richard D.	Minneapolis
§ Grinley, Andrew V.	Grand Rapids
§ Grohs, William H.	Duluth
§ Haase, Donald D.	Silver Bay
§ Haavik, John E.	Duluth
Halbert, John J.	Duluth
§ Halliday, Phillip V.	Duluth
§ Halper, Bernard	Hibbing
Halverson, Kermit J.	Chisholm
† Haney, Claude L.	Duluth
§ Hansen, Robert E.	Hibbing
§ Hanson, Ernest O.	Cloquet
§ Harrington, Vernon A., Jr.	Duluth
†§Hatch, Walter E.	Duluth
Heiam, William C.	Cook
§ Hilding, Anderson C.	Duluth
† Hill, Frederick E.	Riverside, Calif.
† Hill, John P.	Lima, Ohio
† Hirschboeck, Frank J.	Duluth
§ Hoeg, Dwight C.	Duluth
§ Hoff, Herbert O.	Duluth
† Hoover, Norman W.	Rochester
§ Houkom, Samuel S.	Duluth
Hutchinson, Henry	Moose Lake
† Indihar, John E., Jr.	Minneapolis
§ Ireland, Gerald W.	Grand Rapids
§ Jacobson, Clarence	Chisholm
§ Jacobson, Ferdinand C.	Duluth
†§Jensen, Thorvold J.	Duluth
§ Jeronimus, Henry J.	Duluth
Jessico, Charles M.	Duluth
§ Joffe, Harold H.	Virginia
John, Robert H.	Duluth
Johnsen, Henry A., Jr.	Edina
§ Johnson, Calvin J.	Grand Rapids
Johnson, Edward A.	Minneapolis
§ Johnson, Karl E.	Duluth
§ Johnsrud, Luverne W.	Hibbing
§ Johnston, Henry W.	Virginia
†§Jolin, Francis M.	Grand Rapids
Jorgenson, Edward O.	Duluth
§ Juntunen, Roy R.	Duluth
§ Karges, Laurel E.	Grand Rapids
§ Kelley, Walter M.	Duluth
§ Kelly, Robert T.	Grand Rapids
†§Klein, Harry	Duluth
§ Klein, William A.	Duluth
§ Knapp, Frank N.	Duluth
§ Knoedler, John P.	Duluth
Knutson, Kenneth R.	Hibbing
§ Kohlbry, Carl O.	Duluth
Kosiak, William	Two Harbors
§ Kotchevar, Frank R.	Eveleth
Kozberg, Oscar	Moose Lake
§ Krueger, Victor R.	Nopeming
Kruger, Elmer L., Jr.	Nashwauk
§ LaBree, Robert H.	Duluth
†§Laird, Arthur T.	Duluth
Larson, Keith D.	Moose Lake
§ Latterell, Kenneth E.	Duluth
§ Law, Harrison E.	Virginia
§ Leek, Joseph H.	Duluth
Lepak, Francis J.	Duluth
Leppo, N. Erkki A.	Duluth
L 'Esperance, Bernard F.	Two Harbors
Linke, Charles A.	Duluth
Linke, Clara L.	Duluth
† Lipinski, Stanley W.	Memphis, Tenn.
§ Litman, Samuel N.	Duluth
† McCoy, Mary K.	Duluth
† McDonald, Archibald L.	Unknown
§ McDonald, Owen G.	Duluth
McGill, Leon K.	Virginia
§ McHaffie, Orval L.	Duluth
§ McKenna, John J.	Virginia
§ McKenna, Maurice J.	Grand Rapids
§ McNutt, John R.	Duluth
§ MacRae, Gordon C.	Duluth
§ Magney, Fredolph H.	Duluth
§ Malmstrom, John A.	Virginia
§ Marrone, Patrick H.	Duluth
§ Martin, Webster C.	Duluth
§ Martin, William B.	Duluth
§ Mast, Fredric L.	Chisholm
§ Mayne, Roy M.	Nopeming
† Mead, Charles H.	Duluth
*§Merriman, Lloyd L.	Duluth
§ Miettunen, John B.	Hibbing
Milroy, Thomas W.	Des Moines, Iowa
Moe, Thomas	Moose Lake
§ Moehring, Henry G.	Duluth
Mollers, Theodore P.	Soudan
§ Monserud, Nels O.	Cloquet
§ Morseman, L. William	Hibbing
§ Moyer, John B.	Duluth
Muller, John J.	Hibbing
Munger, James E.	Duluth
Munson, Martin S.	Barnum
§ Murray, Robert A.	Hibbing
Nakamura, James Y.	Deer River
§ Neff, Walter S.	Virginia
†§Nicholson, Murdoch A.	Duluth
§ Nisius, George F.	Duluth
§ Nollet, Donald J.	Hibbing
§ Norberg, Carl E.	Cloquet
§ Nutting, Roland E.	Duluth
Nyhus, Ralph E.	Hibbing
§ O'Neill, John C.	Duluth
§ Olson, Albert E.	Duluth
§ Olson, Archie O.	Duluth
Owens, Ben P.	Hibbing
Paciotti, Vincent J.	Hibbing
§ Papermaster, Ralph	Two Harbors
§ Parker, Wilbert H.	Chisholm
§ Parson, E. Irvine	Duluth
§ Pasek, Antone W.	Cloquet
§ Patch, Orien B.	Duluth
Payne, Richard E.	Virginia
† Pearsall, R. P.	Virginia
§ Pedersen, Roy C.	Duluth
† Pennie, Daniel F. V.	Duluth
Peterson, Edward N.	Virginia
§ Peterson, John H.	Duluth
§ Pierce, Jack R.	Williamson, W. Va.
§ Pollard, William S.	Duluth
§ Power, John E.	Duluth
§ Power, John E., Jr.	Duluth
Prlina, Isaac M.	Virginia
Puumala, Reino H.	Cloquet
Raattama, John W.	Nashwauk
Rajala, Arnold I.	Grand Rapids
§ Reardon, Andrew E.	Duluth
Reed, Henry H.	Duluth
§ Reed, Paul	Virginia
§ Relchelt, Deland G.	Duluth
§ Richter, David J.	Virginia
Rondeau, Eugene T.	Two Harbors
† Rowe, Olin W.	Duluth
§ Rowles, Everett K.	Coleraine
§ Rudie, Peter S.	Duluth
§ Rudie, William D.	Duluth
§ Runquist, John M.	Duluth
Ryan, William J.	Duluth
§ Sach-Rowitz, Alan	Moose Lake
§ Salter, Reginald A.	Virginia
Sanford, John B.	Duluth
†§Sarff, Oliver E.	Duluth
Sax, Milton H.	Duluth
Sax, Simon G.	Duluth
§ Schirber, Martin J.	Grand Rapids
§ Schmid, John F.	Duluth
§ Schneider, Lawrence E.	Duluth
† Schroder, Charles H.	Pottstown, Penn.
§ Schweiger, Theodore R.	Hibbing
§ Seashore, Rosel T.	Duluth
Sekanina, Jan	Babbitt
§ Sher, David A.	Virginia
§ Siegel, John S.	Virginia
§ Sinamark, Andrew	Hibbing
§ Sisler, Clifford E.	Minneapolis
§ Smith, Cyril M.	Duluth
Smith, Wallace R.	Grand Marais
Snyker, Omer E.	Ely
§ Soderberg, Richard J.	Grand Marais
§ Spang, Anthony J.	Duluth
§ Spang, James S.	Duluth
§ Spang, William M.	Duluth
§ Stein, William A.	Duluth
§ Storsteen, Kenneth A.	Duluth
† Strathern, Moses L.	Gilbert
§ Streitz, John M.	Duluth
§ Strewler, Gordon J.	Duluth
Summar, M. Thomas	Virginia
§ Sutherland, Harry N.	Ely
§ Swedberg, William A.	Duluth
§ Swenson, Arnold O.	Duluth
§ Swenson, Richard W.	Hibbing
§ Teich, Kenneth W.	Duluth
§ Terrell, Bernard J.	Nopeming
§ Tetlie, James P.	Duluth
§ Thomas, John V.	Duluth
§ Thouin, Laurence G.	Hibbing
§ Tomhave, Wesley G.	Hibbing
§ Tosseland, Noel E.	Duluth
§ Trautmann, James C.	Duluth
† Tuohy, Edward L.	Santa Barbara, Calif.
§ Tuura, James L.	Duluth
*§Urberg, Sofus E.	Duluth
Van Ryzin, Donald J.	Duluth
§ Walder, Harold J.	Duluth
† Walker, Alfred E.	St. Paul
§ Wallace, Martin O.	Duluth
§ Walter, Frederick H.	International Falls
Weir, Matthew J.	Virginia
§ Wells, Arthur H.	Duluth
†§Wheeler, Daniel W.	Duluth
Wilbur, Oscar M.	Hibbing
§ Williams, Bruce F. P.	Duluth
† Winter, John A.	Duluth
†§Wolff, John M.	Duluth
Woodruff, Whitney	Virginia
§ Young, Thomas O.	Duluth
§ Zemmers, Roberts	Duluth

SCOTT-CARVER COUNTY MEDICAL SOCIETY

Regular meetings, third Wednesday of every month, except July and August.
Annual meeting, third Wednesday in June
Number of Members—33

President	
KUCERA, STANLEY T.	Northfield
Secretary	
PHILP, DAVID R.	Watertown
§ Bean, Charles N.	Waconia
Bratholdt, James W.	Watertown
† Buck, Frederick H.	Shakopee
§ Cervenka, Charles F.	New Prague
§ Clarke, John W.	Waconia
§ Doherty, Elmer M.	New Prague
Gildersleeve, Robert	Watertown
†§Hebeisen, Milton B.	Carver
Heinz, Ivy B.	Shakopee
Heinz, Lawrence H.	Shakopee
§ Heinzerling, Carl R.	Chaska
§ Juergens, Herman M.	Belle Plaine
§ Kucera, Stanley T.	Northfield
Larson, Leighton W.	Waconia
§ Lukk, Olaf	Montgomery
† Martin, Thomas Philip	Minneapolis
Nagel, Harold D.	Minneapolis
Ninneman, Newton N.	Waconia
† Novak, Edward E.	New Prague
Olson, Chester J.	Belle Plaine
§ Pearson, Bror F.	Shakopee
§ Philp, David R.	Watertown
* Pogue, Richard E.	Carson City, Nevada
§ Ponterio, James E.	Shakopee
Rieschl, Elizabeth K.	Jordan
§ Rynda, Edwin R.	New Prague
§ Sawaryniuk, Iwan	Waconia
Schimelpfenig, George T.	Chaska
Simmonds, Harry N.	Prior Lake
§ Simons, Bernard H.	Chaska
Stahler, Paul A.	Jordan
†§Westerman, Alvin	Montgomery
†§Westerman, Fred C.	Montgomery

SOUTHWESTERN MINNESOTA MEDICAL SOCIETY

Cottonwood, Jackson, Murray, Nobles, Pipestone, and Rock Counties
Regular meetings, Spring and Fall. Annual meeting, October
Number of Members—61

President	
LAIKOLA, LESLIE A.	Adrian
Secretary	
HEIBERG, OLAF M.	Worthington
§ Arnold, Elmer W.	Adrian
§ Bader, J. L.	Slayton
§ Basinger, Harold P.	Windom
§ Basinger, Homer P.	Windom
§ Beckering, Gerrit	Edgerton
§ Benjamin, Walter G.	Pipestone
§ Bofenkamp, F. William	Luverne
§ Boone, Ervin S.	Luverne
Boyd, Frank E.	Jasper
‡ Buresh, Kenneth L.	Unknown
§ Carlson, John V.	Westbrook
§ Christiansen, Harold A.	Jackson
§ Dawson, Lorin D.	Worthington
Dokken, James H.	Windom
Doman, Victor W.	Lakefield
Hallin, Roger P.	Worthington
§ Halloran, Walter H.	Jackson
§ Halpern, David J.	Brewster
Harada, Thomas T.	Lake Wilson
§ Harrison, Percy W.	Worthington
§ Heiberg, Olaf M.	Worthington
§ Hoyer, Ludolf J.	Windom
§ Karleen, Bernard N.	Jackson
§ Keyes, Robert W.	Pipestone
§ Kilbride, Edwin A.	Worthington
§ Koenecke, F. H.	Lakefield
§ Kotval, Russell J.	Pipestone
§ Laikola, Leslie A.	Adrian
§ Lohmann, John G.	Pipestone
§ Maitland, Edwin T.	Jackson
† Manson, Frank M.	Worthington
§ Martin, Albert C.	Luverne
§ Minge, Raymond K.	Worthington
§ Nealy, Donald E.	Adrian
§ Nicholson, Richard W.	Heron Lake
§ Nywall, Dean D.	Slayton
§ Odland, Donald M.	Luverne
†§Pankratz, Peter J.	Taiwan (Formosa)
§ Patterson, Hugh D.	Slayton
§ Pierson, Roy F.	Slayton
†§Piper, William A.	Mountain Lake
Plott, Carol L.	Heron Lake
§ Plucker, Milton W.	Worthington
‡ Ritzinger, Frederick R.	Gowerie, Iowa
§ Robinett, Robert W.	Worthington
§ Rohrer, Christian A.	Worthington
§ Rose, John T.	Lakefield
Ryding, Vincent	Mountain Lake
§ Sawtell, Robert R.	Worthington
§ Schade, Frederick L.	Worthington
§ Schutz, Elmer S.	Mountain Lake
§ Sherman, Charles L.	Luverne
§ Slater, Sidney A.	Worthington
*†§Sogge, Ludwig L.	Windom
§ Stam, John	Worthington
Stevenson, Basil M.	Fulda
§ Stratte, Harold C.	Windom
§ Vix, Vernon A.	Worthington
§ Wells, Walter B.	Jackson
† Williams, Charles A.	Pipestone
† Williams, Leon A.	Minneapolis
‡ Zeller, Nichols H.	Unknown

STEARNS-BENTON COUNTY MEDICAL SOCIETY

Stearns and Benton Counties
Regular meetings, third Thursday of month. Annual meeting, December
Number of Members—71

President	
BEUNING, JOHN B.	St. Cloud
Secretary	
PETERSEN, ROBERT T.	St. Cloud
§ Alden, W. Charles	Kimball
§ Anderson, Edward M.	St. Cloud
§ Autrey, William A.	St. Cloud
§ Baumgartner, Florian H.	Albany
§ Beuning, John B.	St. Cloud
§ Brigham, Charles F., Jr.	St. Cloud
§ Broker, Henry M.	St. Cloud
§ Buscher, Julius C.	St. Cloud
§ Cesnik, Robert J.	Sauk Rapids
§ Clark, Harry B.	St. Cloud
Cleaves, William D.	Sauk Centre
§ Davis, Arthur E., Jr.	St. Cloud
Davidson, William D.	St. Joseph
§ Donaldson, Charles S.	St. Cloud
† DuBois, Julian F.	Sauk Centre
DuBois, Julian F., Jr.	Sauk Centre
§ Evans, Leslie M.	Sauk Rapids
Fidelman, Norman E.	Foley
†§Fleming, Thomas N.	St. Cloud
§ Gaida, Joseph B.	St. Cloud
§ Goehrs, Gilman H.	St. Cloud
† Goehrs, Henry W.	St. Cloud
Grant, John C.	Sauk Centre
† Haberman, Emil	Osakis
Halenbeck, Philip L.	St. Cloud
Hedlund, Charles J.	St. Paul
Henry, Clarence J.	Milaca
Henry, Joseph E.	Milaca
†§Jones, Richard N.	St. Cloud
Keith, Paul J.	Milaca
§ Kelly, John F.	Cold Spring
Kim, Mark K.	St. Cloud
† Kohler, Delphin W.	Tacoma, Wash.
§ Koop, Herman E.	Cold Spring
‡ Koop, Severin H., Jr.	Sauk Rapids
Kuhlmann, Lawrence B.	Melrose
§ LaFond, Edward M.	St. Cloud
Lindeman, Raymond J.	Paynesville
§ Loes, Louis A.	St. Cloud
§ Luckemeyer, Carl J.	St. Cloud
†§McDowell, John P.	St. Cloud
† Meyer, Anthony A.	Minneapolis
§ Milhaupt, Emmett N.	St. Cloud
Mueller, Rudolph B.	Richmond
Murn, Thomas G.	St. Cloud
§ Musachio, Nicholas F.	Foley
Myre, Clifford R.	Paynesville
§ Neils, Vernon E.	Sauk Rapids
§ Nessa, Curtis B.	St. Cloud
Nietfield, Aloys	Sauk Centre
§ O'Keefe, James P.	St. Cloud
§ Olinger, John N.	St. Cloud
§ Petersen, Robert T.	St. Cloud
§ Phares, Otto C.	St. Cloud
§ Raetz, Sylvester J.	Maple Lake
§ Reif, Henry J.	St. Cloud
§ Richards, William B.	St. Cloud
Salk, Richard J.	Albany
†§Sandven, Nels O.	Paynesville
*†Schatz, Francis J.	St. Cloud
§ Schmitz, Everett J.	St. Cloud
*†Sherwood, George E.	Kimball
§ Sisk, Harvey E.	St. Cloud
† Stangl, Philip E.	St. Cloud
§ Thuringer, Carl B.	St. Cloud
Vanderpool, Thomas E.	Paynesville
Veranth, Leonard A.	St. Cloud
§ Walfred, Karl A.	St. Cloud
§ Wenner, Waldemar T.	St. Cloud
§ Wittrock, Louis H.	Watkins
Zachman, Albert H.	Melrose

STEELE COUNTY MEDICAL SOCIETY

Steele County
Regular meetings, third Tuesday of every other month. Annual meeting, January
Number of Members—24

President	
HENRY, KENNETH G.	Owatonna
Secretary	
FLOERSCH, ADRIAN J.	Owatonna
§ Anderson, Franklin C.	Owatonna
§ Arnesen, John F.	Owatonna
§ Dewey, Donald H.	Owatonna
*†Ertel, Edward Q.	Ellendale
§ Fischer, John R.	Blooming Prairie
§ Floersch, Adrian J.	Owatonna
§ Halvorsen, Daniel K.	Owatonna
Hartung, Elmer H.	Claremont
§ Henry, Kenneth G.	Owatonna
§ Honath, Donald H.	Owatonna
§ Huff, J. S.	Owatonna
Kulstad, Oscar S.	Dodge Center
‡ Kurtin, Joseph J.	New York, N. Y.
§ Lundquist, Curt W.	Owatonna
§ McEnaney, Clifford T.	Owatonna
§ McIntyre, John A.	Owatonna
†§Melby, Benedik	Blooming Prairie
† Morehead, Dewey E.	Owatonna
§ Olson, Albert J.	Owatonna
§ Roberts, Oliver W.	Owatonna
† Senn, Edward W.	Owatonna
§ Schaefer, Joseph F.	Owatonna
§ Stransky, Theodore W.	Owatonna
†§Wilkowske, Rudolph J.	Owatonna

UPPER MISSISSIPPI MEDICAL SOCIETY

Aikin, Beltrami, Cass, Clearwater, Crow Wing, Hubbard, Koochiching,
Lake of the Woods, Morrison, Todd and Wadena Counties
Annual Meeting, January
Number of Members—105

President
Grose, Frederick N. .... Clarissa

Secretary
Badeaux, George I. .... Brainerd

§ Adkins, James J. .... Bertha
§ Aga, John H. .... Brainerd
§ Anderson, Arden O. .... Brainerd
§ Anderson, Werner W. .... Brainerd
Badeaux, George I. .... Brainerd
Belcher, R. A. .... Little Falls
§ Bender, James H. .... Brainerd
§ Benson, Alfred H. .... Little Falls
§ Bissinger, Lester L. .... Brainerd
§ Bolstad, Owen C. .... Little Falls
§ Borgerson, Arthur H. .... Long Prairie
Brennan, Joseph G. .... Browerville
Burman, Richard E. .... Aitkin
Byrne, William J. .... St. Paul
§ Cardle, George E. .... Brainerd
‡ Carlson, Robert G. .... Seattle, Wash.
Closuit, Frederick C. .... Aitkin
Cook, Jay M. .... Staples
§ Coombs, Carl H. .... Cass Lake
Craig, Clair C. .... International Falls
Cushing, Robert L. .... Brainerd
Davis, Lloyd T. .... Wadena
Davis, Luther F. .... Wadena
Deweese, Wilford J. .... Bemidji
Dillenburg, C. J. .... Little Falls
§ Dodson, Albertus F. .... Brainerd
§ Eiler, John .... Park Rapids
§ Erickson, Alvin O. .... Long Prairie
§ Fitzsimons, William E., Jr. .... Brainerd
Fortier, George M. A. .... Little Falls
Garlock, DeWitt W. .... Highlands, Calif.
† Ghostley, Mary C. .... Santa Ana, Calif.
*†Gilmore, Rowland .... Puposky
Griffin, John W., Jr. .... Bemidji
Grimes, Paul T. .... Park Rapids
Groschupf, Richard P. .... Bemidji
Groschupf, Theodore P. .... Bemidji
§ Grose, Frederick N. .... Clarissa
Halme, William H. .... Wadena
§ Hanover, Ralph D. .... International Falls
Hansen, Milo L. .... Little Falls
Hartjen, Jason K. .... Bemidji
† Healy, Raymond T. .... Pierz
Heid, James K. .... Little Falls
Heinz, John W. .... Wadena
Hendricks, Esten J. .... Verndale
Hildebrand, John E. .... Bemidji
Hoganson, Donald E. .... Bemidji
* Houston, Donald M. .... Park Rapids
§ Hughes, Bernard J. .... Brainerd
Johnson, Douglas L. .... Little Falls
† Johnson, Einar W. .... Bemidji
§ Kanne, Earl R. .... Brainerd
Kelley, Roger E. .... Crosby
Kinports, Edward B. .... International Falls
§ Knoll, W. V. .... Brainerd
Larson, LeRoy J. .... Bagley
Lee, Hubert W. .... Brainerd
† Lenarz, Albert .... Browerville
§ Lofstrom, Dennis E. .... Pine River
Longfellow, Helen W. .... Brainerd
Lund, Werner J. .... Staples
Lundsten, Leslie C. .... Bemidji
§ McLane, William O. .... Brainerd
§ Marshall, Clark M. .... Crosby
§ Marvin, Joseph E. .... Brainerd
§ Meller, Maurice .... Brainerd
§ Mortenson, Howard O. .... Menahga
§ Mosby, Maurice E. .... Long Prairie
§ Mulligan, Arthur M. .... Brainerd
Murtaugh, Robert J. .... Wadena
*†Nelson, Nesmith P. .... Minneapolis
Nichols, R. D. .... Crosby
Nixon, James B. .... Crosby
Nord, J. Erling .... Staples
Nordlund, Mildred E. .... Cass Lake
§ O'Leary, John B. .... Brainerd
Olson, Lillian A. .... Ah-gwah-ching
Palmer, Harry A. .... Black Duck
Parker, Charles W. .... Wadena
Parker, Philip J. .... Staples
§ Parker, Warren E. .... Sebeka
§ Pedersen, Robert L. .... Brainerd
Pelzl, Charles R. .... Pine River
Pettersen, George R. .... Aitkin
Pierce, Charles H. .... Wadena
Quanstrom, Virgil E. .... Brainerd
§ Reichelderfer, Charles F. .... Staples
§ Ringle, Otto F. .... Walker
† Sanderson, Anton G. .... Ashby
Simons, Edwin J. .... Edina
§ Skaife, William F. .... Little Falls
Spieler, Forrest B. .... Pequot Lakes
§ Stein, Raymond J. .... Pierz
§ Stoy, Robert A. .... Little Falls
Ulrich, Emery E. .... Crosby
Vandersluis, Charles W. .... Bemidji
§ Watson, Alexander M. .... Royalton
† Watson, Percy T. .... Miami, Fla.
§ Watson, Sidney W. .... Royalton
Whittemore, Dexter D. .... Bemidji
§ Will, Charles B. .... International Falls
Will, W. W. .... Bertha
§ Williams, Mervyn M. .... Ah-gwah-ching
Witter, Robert L. .... Wadena

WABASHA COUNTY MEDICAL SOCIETY

Wabasha County
Regular meeting, special call. Annual meeting, first Thursday after first Monday in October
Number of Members—14

President
Sontag, David W. .... Lake City

Secretary
Flesche, Bernard A. .... Lake City

§ Bayley, E. Covell .... Lake City
§ Bouquet, Bertram J. .... Wabasha
§ Bowers, Robert N. .... Lake City
† Collins, Joseph S. .... Wabasha
§ Ekstrand, LeRoy M. .... Wabasha
§ Ellis, Earl Wm. .... Elgin
§ Flatt, John R. .... Wabasha
§ Flesche, Bernard A. .... Lake City
§ Gjerde, William P. .... Lake City
Glabe, Robert A. .... Plainview
§ Mahle, Donald G. .... Plainview
§ Ochsner, Clarence G. .... Wabasha
† Replogle, William H. .... Los Angeles, Calif.
§ Sontag, David W. .... Lake City

WASECA COUNTY MEDICAL SOCIETY

Waseca County
Regular Meetings, January. Annual Meetings, January.
Number of Members—8

President
Olds, George H. .... New Richland

Secretary
Florine, Martin C. .... Janesville

§ Davis, Raymond D. .... Waseca
§ Florine, Martin C. .... Janesville
†§Gallagher, Bernard J. .... Waseca
§ Hottinger, Raymond C. .... Janesville
§ Normann, Stephen T., Jr. .... Waseca
§ Oeljen, Siegfried C. G. .... Waseca
§ Olds, George H. .... New Richland
§ Swenson, Orvie J. .... Waseca

WASHINGTON COUNTY MEDICAL SOCIETY

Washington and Dakota Counties
Regular meetings, second Tuesday in each month except June, July and August
Annual meeting, second Tuesday in December
Number of members—23

President
Carlson, Russel E. .... Stillwater

Secretary
Kiolbasa, Edward B. .... Stillwater

Brabec, Paul F. .... Hastings
§ Carlson, Russel E. .... Stillwater
Chappell, Elliott R. .... Stillwater
Fasbender, Herman T. .... Hastings
†§Holcomb, Joel T. .... Marine-on-St. Croix
† Humphrey, Wade R. .... Stillwater
§ Jenson, James E. .... Stillwater
§ Johnson, Robert H. .... Chisago City
Josewski, Raymond J. .... Stillwater
§ Juergens, Manley F. .... Stillwater
Just, Herman J. .... Hastings
§ Kiolbasa, Edward B. .... Stillwater
Kulzer, Norbert J. .... Hastings
§ McCarten, Francis M. .... Stillwater
Mensheha, Nicholas .... Forest Lake
† Poirier, Joseph A. .... Forest Lake
Ruggles, George M. .... Forest Lake
§ Sherman, Carnot H. .... Bayport
§ Stuhr, John W. .... Stillwater
Torghele, John R. .... Hastings
Van Meier, Henry .... Stillwater
Weiss, Carl A. .... Hastings
Wood, Lloyd T. .... Forest Lake

WEST CENTRAL MINNESOTA MEDICAL SOCIETY

Big Stone, Pope, Stevens, and Traverse Counties

Regular meetings, one Tuesday in March, May, September and November
Annual meeting, November

Number of Members—29

President	
OLIVER, IRWIN L.	Graceville
Secretary	
SWENDSEEN, CARL J.	Graceville
§ Arneson, Arthur I.	Morris
§ Behmler, Frederick Wm.	Morris
†§Bergan, Otto	Clinton
† Bolsta, Charles	Ortonville
§ Bucher, Foster D.	Starbuck
†§Eberlin, Edward A.	Glenwood
§ Eide, O. A.	Hancock
† Elsey, James R.	Glenwood
Good, Roy H.	Glenwood
Hedemark, Homer H.	Ortonville
Hedemark, Truman A.	Ortonville
Karn, Jacob F.	Ortonville
Kolp, Berton A.	Glenwood
§ Kooda, Jennings C.	Morris
Lee, Gordon E.	Glenwood
Letson, Robert D.	Glenwood
§ Lindberg, Alfred L.	Wheaton
†§Linde, Herman	Cyrus
§ Magnuson, Allen E.	Wheaton
§ Merrill, Robert W.	Morris
§ Muir, Walter E.	Browns Valley
§ Oliver, Irwin L.	Graceville
† Ranson, Matthias L.	Hancock
§ Rossherg, Raymond A.	Morris
§ Swedenburg, Paul A.	Glenwood
Swendseen, Carl J.	Graceville
§ Watson, Robert M.	Morris
Winge, H. C.	Wheaton
Zempel, Alan R.	Starbuck

WINONA COUNTY MEDICAL SOCIETY

Winona County

Regular meetings, first Monday in January, April, July and September
Annual meeting, first Monday in January

Number of Members—34

President	
HEISE, CARL vR.	Winona
Secretary	
JOHNSON, CURTIS M.	Winona
§ Boardman, Dalmon V.	Winona
§ Christensen, Eli E.	Winona
Degallier, Daniel	Winona
§ Finkelnburg, William O.	Winona
§ Haesly, Warren W.	Winona
§ Hartwich, Roger F.	Winona
Hawk, Dale J.	St. Charles
§ Heise, Carl vR.	Winona
§ Heise, Herbert vR.	Winona
§ Heise, Paul vR.	Winona
§ Heise, Philip vR.	Winona
§ Hughes, Sidney O.	Winona
§ Johnson, Curtis M.	Winona
§ Johnston, Leonard F.	Winona
§ Keyes, John D.	Winona
§ Loomis, George L.	Winona
§ McHutchison, Samuel	St. Charles
†§McLaughlin, Edmund M.	Winona
§ Mattison, Percy A.	Winona
§ Meinert, Albert E.	Winona
Neumann, Conrad A.	Lewiston
§ Page, Raymond L.	St. Charles
†§Robbins, Charles P.	Winona
§ Roemer, Henry J.	Winona
§ Rogers, Charles W.	Winona
§ Satterlee, Howard W.	Lewiston
§ Schmidt, Hilmar R.	Rushford
§ Testor, James V.	Winona
§ Tweedy, John A.	Winona
§ Tweedy, Robert B.	Winona
§ Vollmer, Frederick J.	Winona
§ Wilson, Louis J.	Winona
§ Wilson, Rolland H.	Winona
§ Younger, Lewis I.	Winona

WRIGHT COUNTY MEDICAL SOCIETY

Wright County

Regular meetings, first Tuesday of every second month.
Annual meeting, in November

Number of Members—16

President	
PURVES, G. HARLAND	Buffalo
Secretary	
CATLIN, THEODORE J.	Buffalo
§ Anderson, Waldo P.	Buffalo
§ Bendix, Lester H.	Annandale
†§Catlin, John J.	Buffalo
§ Catlin, Theodore J.	Buffalo
§ Ellison, Frank E.	Monticello
§ Greenfield, William T.	Cokato
* Grundset, Ole J.	Montrose
§ Guilfoile, Pierre J.	Delano
§ Hall, William E.	Maple Lake
§ Hart, William E.	Monticello
§ Purves, G. Harland	Buffalo
§ Sandeen, Robert M.	Buffalo
§ Smorstok, Matthew B.	Monticello
§ Thielen, Robert D.	Saint Michael
§ Thomas, William H.	Howard Lake
§ Vaaler, Robert T.	Cokato

ZUMBRO VALLEY MEDICAL SOCIETY

Olmsted, Houston, Fillmore and Dodge Counties

Regular meetings, 1st Wednesday of odd numbered months.
Annual meeting, 1st Wednesday in November.

Number of Members—579

President	
WILSON, VIKTOR O.	Rochester
Secretary	
WELLMAN, WILLIAM E.	Rochester
§ Aaro, Leonard A.	Rochester
§ Achor, Richard W. P.	Rochester
† Adams, Ralph E.	Rochester
† Addison, Robert M.	Rochester
§ Adson, Martin A.	Rochester
Affeldt, Daniel E.	Kasson
§ Ahlfs, Jacob J.	Caledonia
† Allaman, Loren E.	Rochester
§ Allen, Edgar V. N.	Rochester
† Amberg, Samuel	Rochester
§ Andersen, Howard A.	Rochester
§ Anderson, Mark J.	Rochester
§ Anderson, Markham J., Jr.	Rochester
§ Anderson, Milton W.	Rochester
† Arnold, John W.	Rochester
† Anzel, Sanford H.	Rochester
Baars, Conrad W.	Rochester
§ Baggenstoss, Archie H.	Rochester
§ Bair, Hugo L.	Rochester
§ Baker, George S.	Rochester
† Baker, Harry R.	Hayfield
§ Baker, Hillier L., Jr.	Rochester
†§Balfour, Donald C.	Rochester
Balfour, William M.	Lawrence, Kans.
Balz, George P.	Toledo, Ohio
§ Banner, Edward A.	Rochester
§ Bargen, J. Arnold	Rochester
§ Barker, Nelson W.	Rochester
†§Barnes, Arlie R.	Rochester
§ Barry, Maurice J., Jr.	Rochester
§ Bartholomew, Lloyd G.	Rochester
§ Bastron, James A.	Rochester
§ Bayrd, Edwin D.	Rochester
§ Beahrs, Oliver H.	Rochester
† Beargie, Richard J.	Rochester
†§Benedict, William L.	Rochester
§ Berge, Kenneth G.	Rochester
*§Berkman, David M.	Oronoco
§ Berkman, John M.	Rochester
† Berman, Harold	Rochester
† Berman, Irwin B.	Rochester
† Berman, Stanley	Rochester
§ Bernatz, Philip E.	Rochester
§ Bickel, William H.	Rochester
† Bigelow, Charles E.	Dodge Center
† Bigg, Richard L.	Rochester
† Billingsley, John S.	Rochester
Birkhead, Newton C.	Rochester
§ Black, B. Marden	Rochester
§ Blackburn, Charles M.	Rochester
Blakey, Leslie W.	Rochester
† Bledsoe, Francis H.	Rochester
Bonnet, John D.	Rochester
† Boolukos, George P. N.	Rochester
† Bowen, Ralph, Jr.	Rochester
§ Boyd, David A., Jr.	Rochester
Boyd, George K.	Rochester
†§Braasch, William F.	Rochester
§ Brandenburg, Robert O.	Rochester
§ Broadbent, James C.	Rochester
Brow, Raymond E.	Rochester

§ Brown, Alexander E. .... Rochester
§ Brown, Henry A. .... Rochester
§ Brown, Joe R. .... Rochester
§ Brown, Philip W. .... Rochester
§ Brunsting, Louis A. .... Rochester
† Buie, Louis A. .... Rochester
§ Burchell, Howard B. .... Rochester
Burger, Thomas C. .... Evansville, Ind.
Burgert, E. O., Jr. .... Rochester
§ Burich, Harry F. .... Rochester
§ Burke, Edmund C. .... Rochester
§ Butt, Hugh R. .... Rochester
† Cadman, Norman L. .... Rochester
§ Cain, James C. .... Rochester
§ Callahan, John A. .... Rochester
† Calverley, John R. .... Rochester
§ Campbell, Donald C. .... Rochester
§ Campbell, Malcolm K. .... Rochester
† Carnevali, John F. .... Rochester
Carr, David T. .... Rochester
§ Carryer, Haddon McC. .... Rochester
Cawein, Madison .... Rochester
§ Childs, Donald S., Jr. .... Rochester
§ Christensen, Norman A. .... Rochester
§ Clagett, O. Theron .... Rochester
§ Clark, Edward C. .... Rochester
§ Clark, Leslie W. .... Spring Valley
† Clifton, Theodore A. .... Hollywood, Fla.
Climie, Andrew R. .... Rochester
‡ Clowdus, Bernard F. ....
Bolling Air Force Base, D. C.
† Coffey, William F. X. .... Rochester
§ Colby, Malcolm Y., Jr. .... Rochester
*§Comfort, Mandred W. .... Rochester
§ Connolly, Daniel C. .... Rochester
† Connor, P. James .... Rochester
§ Cook, Edward N. .... Rochester
§ Cooper, Talbert .... Rochester
§ Corbin, Kendall B. .... Rochester
† Corr, William P. .... Rochester
Corrigan, Cyril J. .... Rochester
‡ Cottone, Francis J. .... Trenton, New Jersey
Courtright, Claiborne .... Rochester
§ Coventry, Markham B. .... Rochester
Covey, Kenneth W. .... Rochester
†§Craig, Winchell McK. .... Rochester
§ Culp, Ormond S. .... Rochester
§ Dahlin, David C. .... Rochester
§ Daly, David D. .... Rochester
Danford, Harold G. .... Appleton, Wis.
§ Daugherty, Guy W. .... Rochester
† Davidson, Paul .... Rochester
† Davis, Austin C. .... Florida
§ Davis, George D. .... Rochester
§ Dearing, William H. .... Rochester
Decker, Barry .... Rochester
§ Decker, David G. .... Rochester
§ Devine, Kenneth D. .... Rochester
§ DeWeerd, James H. .... Rochester
§ Deissner, Grant R. .... Rochester
Divertie, Matthew .... Rochester
§ Dixon, Claude F. .... Rochester
† Dobbs, Richard L. .... Rochester
§ Dockerty, Malcolm B. .... Rochester
§ Dodge, Henry W., Jr. .... Rochester
† Dombrowski, Edmond T. .... Rochester
§ Donoghue, Francis E. .... Rochester
§ Douglass, Bruce E. .... Rochester
§ Doyle, James R. .... Rochester
† Drips, Della G. .... Oronoco
Dry, Thomas J. .... Cape Town, Africa
Dunitz, Norman L. .... Rochester
§ DuShane, James W. .... Rochester
§ Dyer, John Allen .... Rochester
§ Eaton, Lealdes M. .... Rochester
Edelmann, Robert B. .... Short Hills, N. J.
† Edelstein, Jack P. .... Rochester
§ Edwards, Jesse E. .... Rochester
Edwin, Russell L. .... Rochester
§ Elkins, Earl C. .... Rochester
§ Ellis, F. Henry .... Rochester
§ Emmett, John L. .... Rochester
Engler, Robert S. .... Rochester
Englund, Garth W. .... Mabel
§ Erich, John B. .... Rochester
§ Erickson, Donald J. .... Rochester
§‡Esser, Robert A. .... Paris, France
§ Estes, J. Earle .... Rochester
†§Eusterman, George B. .... Rochester
Evans, Harold W. .... Grand Forks, N. D.
† Evarts, Arrah B. .... Rochester
† Evenson, David J. .... Rochester
Ewen, Edgar F. .... Rochester
§ Faber, John E. .... Rochester
Failor, Harlan J. .... Rochester
§ Faucett, Robert L. .... Rochester
§ Faulconer, Albert, Jr. .... Rochester
Feinberg, Walter D. .... Rochester
‡ Feinerman, Burton .... Brooklyn, N. Y.
Feldmann, Floyd M. .... New York, N. Y.
§ Ferris, Deward O. .... Rochester
† Figi, Frederick A. .... Rochester
Foss, Edward L. .... Rochester
§ Foulk, William T., Jr. .... Rochester
† Franckowiak, John J. .... Rochester
§ Frazier, Shervert H., Jr. .... Rochester
Frethem, Allen A. .... Rochester
§ Fricke, Robert E. .... Rochester
†§Gambill, Carl M. .... Rochester
§ Gambill, Earl E. .... Rochester
Gardner, Gawinn B. .... Rochester
Gardner, John U. .... Rochester
§ Gastineau, Clifford F. .... Rochester
† Gatzke, Laurence D. .... Rochester
§ Geraci, Joseph E. .... Rochester
§ Ghormley, Ralph K. .... Carmel, Calif.
† Giffin, Herbert Z. .... Rochester
Giffin, Mary E. .... Rochester
§ Gifford, R. W., Jr. .... Rochester
† Giles, William F. .... Rochester
‡ Gill, Charles Richard .... Ft. Meade, Md.
† Gilloon, James R. .... Rochester
Gilpin, Charles A., Jr. .... Rochester
Glathe, John P. .... Rochester
‡ Glick, Dallas D. .... San Francisco, Calif.
§ Goldstein, Norman P. .... Rochester
§ Good, C. Allen, Jr. .... Rochester
‡ Gottlieb, Cornelius M. .... Tacoma, Wash.
Gould, Allan B., Jr. .... Rochester
† Graudins, Gunars .... Rochester
§ Green, Paul A. .... Rochester
§ Greene, Laurence F. .... Rochester
§ Grindlay, John H. .... Rochester
Grizzle, Claude O. .... Rochester
§ Grose, John B. .... Rochester
Gyde, Maurice C. .... Rochester
§ Habein, Harold C. .... Rochester
Habein, Harold C., Jr. .... Unknown
§ Hagedorn, Albert B. .... Rochester
† Haines, Samuel F. .... Rochester
§ Hallberg, Olav Erik .... Rochester
†§Hallenbeck, Dorr F. .... LaJolla, Calif.
§ Hallenbeck, George A. .... Rochester
‡ Hamilton, Warren W., Jr. .... Rochester
§ Hanlon, David G. .... Rochester
§ Hanson, Norbert Orrin .... Rochester
§ Hargraves, Malcolm M. .... Rochester
§ Harrington, Stuart W. .... Rochester
§ Harris, Lloyd E. .... Rochester
Harrison, Edgar G., Jr. .... Rochester
†§Hartman, Howard R. .... Rochester
Hartridge, Virginia B. .... Rochester
Hassel, Carl W., Jr. .... Rochester
† Hauser, Harris M. .... Rochester
† Havens, Fred Z. .... Riverside, Calif.
§ Hayles, Alvin B. .... Rochester
§ Heck, Frank J. .... Rochester
Hedrick, William L. .... Rochester
† Heilman, Dorothy M. H. ....
Long Beach, Calif.
Heilman, Fordyce R. .... Rochester
† Helland, John W. .... Spring Grove
†§Helmholz, Henry F. .... Rochester
†§Hempstead, Bert E. .... Rochester
§ Henderson, John W. .... Rochester
§ Henderson, Lowell L. .... Rochester
§ Hepper, Norman G. .... Rochester
† Hewitt, Edith S. .... Rochester
†§Hewitt, Richard M. .... Topeka, Kan.
*†Heyerdale, Oscar C. .... Rochester
§ Higgins, John A. .... Rochester
§ Hill, John Roger .... Rochester
Hill, Richard Woolsey .... Rochester
Himebaugh, Gilbert J. .... Evansville, Ind.
§ Hines, Edgar A., Jr. .... Rochester
§ Hodgson, Corrin H. .... Rochester
§ Hodgson, John R. .... Rochester
Holland, C. R. .... Rochester
§ Hollenhorst, Robert W. .... Rochester
§ Holman, Colin B. .... Rochester
† Hooker, John P. .... Rochester
† Hopkins, Donald M. .... Rochester
† Horton, Bayard T. .... Rochester
Howard, Frank M., Jr. .... Rochester
§ Howell, Llewelyn P. .... Rochester
Howland, Willard J., Jr. .... Rochester
† Huff, James F. .... Rochester
§ Huizenga, Kenneth A. .... Rochester
§ Hunt, Arthur B. .... Rochester
† Hunt, James C. .... Rochester
Hunt, James Calvin .... Rochester
§ Hunter, James S., Jr. .... Rochester
§ Ivins, John C. .... Rochester
Ivy, Horace K. .... Rochester
§ Jackman, Raymond J. .... Rochester
§ Janes, Joseph M. .... Rochester
Johnson, Adelaide McF. .... Rochester
§ Johnson, Carl E. .... Rochester
§ Johnson, Einer W., Jr. .... Rochester
Johnson, Ralph B. .... Lanesboro
‡ Johnson, Ray A. .... New York, N. Y.
Johnson, Victor .... Rochester
Joyce, George L. .... Rochester
§ Judd, Edward S., Jr. .... Rochester
Juergens, John L. .... Rochester
§ Karansky, Stanley .... Phoenix, Ariz.
Kavanaugh, Gerald J. .... Rochester
Kazdan, Martin S. .... Rochester
§ Kearns, Thomas P. .... Rochester
§ Keating, Francis R., Jr. .... Rochester
§ Keith, Haddow M. .... Rochester
†§Keith, Norman M. .... Rochester
§ Kelly, Patrick J. .... Rochester
§ Kennedy, Roger L. J. .... Rochester
§ Kernohan, James W. .... Rochester
§ Kiely, Joseph M. .... Rochester
§ Kierland, Robert R. .... Rochester
§ Kincaid, Owings W. .... Rochester
† King, Harold E. .... Rochester
† Kinzel, Raymond C. .... Rochester
§ Kirby, Thomas J., Jr. .... Rochester
§ Kirklin, John W. .... Rochester
† Kirshen, Robert .... Rochester
Klass, Donald W. .... Rochester
§ Knutson, Lewis A. .... Spring Grove
§ Koelsche, Giles A. .... Rochester
Kragh, Lyle V. .... Rochester
§ Krusen, Frank H. .... Rochester
§ Kvale, Walter F. .... Rochester
§ Lake, Clifford F. .... Rochester
† Lansche, Richard K. .... Rochester
† Larson, Norman E. .... Rochester
† Larson, Robert L. .... Rochester
Lazarte, Jorge A. .... Rochester
Leavitt, Clark T. .... Rochester
*†Leddy, Eugene T. .... Rochester
§ Lee, Robert E. .... Rochester
Lehoux, Louis M. .... Quebec, Canada
† Leigh, John E. .... Rochester
† Lewis, David R. .... Rochester
*†§Lillie, Harold I. .... Rochester
§ Lillie, John C. .... Rochester
Lindquist, Richard H. .... Rochester
§ Lipscomb, Paul R. .... Rochester
§ Litin, Edward M. .... Rochester
Litin, Robert B. .... Rochester
§ Litzow, Thaddeus J. .... Rochester
§ Lofgren, Karl A. .... Rochester
*†§Logan, Archibald H. .... Rochester
§ Logan, George B. .... Rochester
† Lorenc, Ernest .... Rochester
§ Love, J. Grafton .... Rochester
§ Lundy, John S. .... Rochester
Lyons, William S. .... Douglas
§ MacCarty, Collin S. .... Rochester
†§MacCarty, William C. .... Rochester
MacLean, Alexander R. .... Rochester
§ Magath, Thomas B. .... Rochester
† Maher, Frank T. .... Rochester
Maks, Stephen W. .... Rochester
† Malkasian, George D., Jr. .... Rochester
§ Mankin, Harold T. .... Rochester
Marquis, John W. .... Rochester
§ Martens, Theodore G. .... Rochester
§ Martin, Gordon M. .... Rochester
‡ Martin, Maurice J. .... San Juan, P. R.
§ Martin, William J. .... Rochester
† Masson, Duncan M. .... Rochester
† Masson, James C. .... Rochester
§ Masson, James K. .... Rochester
† Mastman, Gary J. .... Rochester
§ Mathieson, Don R. .... Rochester
† Mayberry, William E. .... Rochester
§ Mayne, John G. .... Rochester
§ Mayo, Charles W. .... Rochester
† McAllister, James .... Rochester
§ McBean, James B. .... Rochester
McCammon, Joseph .... Rochester
McClary, George B., Jr. .... Rochester
McClure, Rensselaer W., Jr. ....
Lawrence, Kansas
§ McConahey, William M., Jr. .... Rochester
McCown, Louis K. .... Memphis, Tenn.
† McDonald, Colin C. .... Rochester
§ McDonald, John R. .... Rochester
McGoon, Dwight C. .... Rochester
McIlhany, Mary Lou .... Lantana, Fla.
McKaig, Carle B. .... Pine Island
McKay, Kenneth H. .... Muscatine, Iowa
† McMahan, Robert O. .... Rochester
§ Merritt, Wallace A. .... Rochester
Metzerott, Kirk O. .... Rochester
†§Meyerding, Henry W. .... Rochester
† Miller, Archie W. .... Rochester
§ Miller, Roland D. .... Rochester
§ Miller, Ross H. .... Rochester
§ Millikan, Clark H. .... Rochester
§ Mills, Stephen D. .... Rochester
† Minor, Ralph H. .... Rochester
†§Moersch, Frederick P. .... Rochester
§ Moersch, Herman J. .... Rochester
Moertel, Charles G. .... Rochester
Molnar, George D. .... Rochester
§ Montgomery, Hamilton .... Rochester
Morisaki, Michael M. .... Rochester
§ Morlock, Carl G. .... Rochester
Morrow, George W., Jr. .... Rochester
† Moskowitz, Roland W. .... Rochester
§ Mulder, Donald W. .... Rochester
Mussey, Mary E. .... Rochester
*†Mussey, Robert D. .... Rochester
§ Myers, Thomas T. .... Rochester
Nehring, Jesse P. .... Preston
† Neil, Robert L. .... Rochester

	Neiswanger, Walter E.	Rochester
	Nelson, Gunard A.	Rochester
	Nichols, Donald R.	Rochester
	Nichols, Francis C.	Woodridge, N. J.
†	Niehaus, Karl F.	Rochester
	Nolan, Robert B.	Rochester
	Norris, Neil T.	Caledonia
†	Norstrom, Craig W.	Rochester
†	Nydell, Carl C., Jr.	Rochester
§	Odel, Howard M.	Rochester
†	Okihiro, Michael M.	Rochester
§	Olsen, Arthur M.	Rochester
§	Olson, Ernest A.	Pine Island
	Olson, Grant E.	West Concord
	Onsgard, L. Kenneth	Houston
*§	Osborn, John E.	Rochester
	Osmundson, Philip J.	Rochester
§	Owen, Charles A., Jr.	Rochester
§	Paris, Jaime	Rochester
§	Parker, Harry L.	Rochester
§	Parker, Robert L.	Rochester
	Parkhill, Edith M.	Rochester
§	Parkin, Thomas W.	Rochester
†	Parr, Eugene Q.	Rochester
	Pascuzzi, Chris A.	Rochester
§	Patrick, Robert T.	Rochester
§	Paulson, John A.	Rochester
	Payne, William S.	Rochester
	Pease, Gertrude L.	Rochester
†	Pemberton, John D.	Rochester
§	Perry, Harold	Rochester
	Peterman, Albert F.	Rochester
§	Peters, Gustavus A.	Rochester
§	Petersen, Magnus C.	Rochester
	Peterson, Lowell F. A.	Rochester
	Peterson, Paul A., Jr.	Rochester
‡	Pettet, John R.	Laguna Beach, Calif.
	Phalen, Patrick T.	Rochester
†	Piper, Monte C.	Lacanada, Calif.
§	Polley, Howard F.	Rochester
§	Pool, Thomas L.	Rochester
	Posey, John W.	Rochester
§	Pratt, Joseph Hyde, Jr.	Rochester
§	Prickman, Louis E.	Rochester
§	Priestley, James T.	Rochester
§	Pruitt, Raymond D.	Rochester
§	Pugh, David G.	Rochester
	Purnell, Don C.	Rochester
†	Quetsch, Richard M.	Rochester
	Ragen, Patrick A.	Rochester
§	Ralston, Donald E.	Rochester
	Ramsdell, John Alan	Rochester
§	Randall, Lawrence M.	Rochester
§	Randall, Raymond V.	Rochester
	Redford, John B.	Rochester
‡	Reifsnyder, William Henry, III	Rochester
§	Reitemeier, Richard J.	Rochester
§	ReMine, William H., Jr.	Rochester
	Restall, Charles J.	Rochester
†	Reynolds, William A.	Rochester
§	Risser, Alden F.	Stewartville
†	Roberts, Frank E.	Rochester
§	Robinson, David B.	Rochester
	Robinson, Hugh P.	Rochester
§	Rogne, William G.	Spring Grove
§	Rome, Howard P.	Rochester
§	Rooke, Edward D.	Rochester
	Ross, Griff T.	Rochester
†	Ross, James V.	Rochester
†	Ross, Margaret	Rochester
‡	Roth, Harry Leo	Carlstad, Calif.
§	Rovelstad, Randolph A.	Rochester
§	Rucker, Charles W.	Rochester
	Rushing, Lige B., Jr.	Rochester
§	Rushton, Joseph G.	Rochester
†	Russell, Marion F., Jr.	Rochester
§	Rynearson, Edward H.	Rochester
	Sager, John H.	Rochester
†	Sager, Stanley M.	Rochester
§	Salassa, Robert M.	Rochester
	Sanchez, James J.	Rochester
†§	Sanford, Arthur H.	Rochester
§	Sauer, William G.	Rochester
	Sayre, George P.	Rochester
	Schadt, Daniel C.	Rochester
§	Scanlon, Paul W.	Rochester
	Schaefer, Joseph C.	Rochester
§	Scheifley, Charles H.	Rochester
	Schirger, Alexander	Rochester
§	Schmidt, Herbert W.	Rochester
	Schneider, James A.	Rochester
	Scholl, Virginia M.	Rochester
§	Scholz, Donald A.	Rochester
†	Schwartz, John T.	Rochester
	Scott-Miller, James R.	Rochester
§	Scudamore, Harold H.	Rochester
	Seay, James Elbert, III	Rochester
‡	Sebrechts, Paul	Great Lakes, Illinois
†	Semler, Herbert J.	Rochester
†	Sherrick, Donald W.	Rochester
§	Shick, Richard M.	Rochester
§	Siekert, Robert G.	Rochester
	Silver, Arthur W.	Rochester
‡	Silvis, Stephen E.	San Francisco Calif.
§	Simonton, Kinsey M.	Rochester
	Skaug, Harold M.	Chatfield
†§	Smith, Frederick L.	Rochester
†§	Smith, Harry L.	Rochester
‡	Smith, John H.	Augusta, Georgia
†	Smith, Lester L., Jr.	Rochester
§	Smith, Lucian A.	Rochester
§	Smith, Reginald A.	Rochester
§	Smith, William G.	Rochester
	Smollen, William J.	Rochester
§	Sommerville, Robert L.	Rochester
§	Soule, Edward H.	Rochester
	Spencer, Robert J.	Rochester
	Sperl, Michael P., Jr.	Rochester
§	Spittel, John A., Jr.	Rochester
§	Sprague, Randall G.	Rochester
	Spudis, Edward V.	Rochester
†	Spurgeon, Frederick C.	Rochester
	Starr, Grier F.	Rochester
†	Starr, Jason L.	Rochester
§	Stauffer, Maurice H.	Rochester
§	Steinhilber, Richard M.	Rochester
§	Stickney, J. Minott	Rochester
§	Stillwell, George K.	Rochester
§	Stilwell, George G.	Rochester
†	Stoltze, Cynthia A.	Rochester
†	Stool, Newsom	Rochester
	Storino, Henry E.	Rochester
	Stormont, James R.	Rochester
§	Stroebel, Charles F., Jr.	Rochester
§	Sullivan, Charles R.	Rochester
§	Svien, Hendrik J.	Rochester
§	Symmonds, Richard E.	Rochester
†	Tama, Lawrence	Rochester
†	Taswell, Howard F.	Rochester
§	Theye, Richard A.	Rochester
§	Thompson, Gershom J.	Rochester
†	Thompson, John W., III	Rochester
§	Tillisch, Jan H.	Rochester
‡	Tuffanelli, Denny L.	New York, N. Y.
	Turner, John C., Jr.	Rochester
	Tyler, Donald E.	Rochester
§	Uihlein, Alfred	Rochester
§	Underdahl, Laurentius O.	Rochester
§	Utz, David C.	Rochester
	Utz, Philip H.	La Crescent
	Vance, John W.	Buffalo, N. Y.
§	Van Herik, Martin	Rochester
†	Vastola, Anthony P., Jr.	Rochester
§	Vaughn, Louis D.	Rochester
§	Verby, John E., Jr.	Rochester
†	Viren, Fred K.	Rochester
	Virnig, Hildegard, J.	Caledonia
†	Wagener, Henry P.	Rochester
	Wagoner, James M.	Harmony
	Wakai, Coolidge S.	Honolulu, Hawaii
§	Wakefield, Elmer G.	Rochester
†	Wakim, Khalil G.	Rochester
§	Waldmann, Edward B.	Rochester
§	Walters, Waltman	Rochester
§	Ward, Louis E.	Rochester
†	Warvel, John H., Jr.	Rochester
§	Watkins, Charles H.	Rochester
	Watkins, Charles W.	Vancouver, Wash.
§	Watson, John R.	Rochester
§	Waugh, John M.	Rochester
†	Weaver, W. F.	Rochester
§	Weber, Harry M.	Rochester
§	Weed, Lyle A.	Rochester
§	Weeks, Richard E.	Rochester
§	Weidman, William H.	Rochester
†§	Weir, James F.	Rochester
§	Welch, John S.	Rochester
§	Wellman, William E.	Rochester
	Wellner, Theodore O.	Rochester
§	Wente, Harold A.	Rochester
§	Westrup, John E.	Lanesboro
§	Weyhrauch, William R.	Rochester
§	Whisnant, Jack P.	Rochester
	Whitcomb, Fred F., Jr.	Rochester
†	White, James C.	Rochester
	Wholey, Mark H.	Rochester
†	Wilbur, Dwight Locke, III	Rochester
†	Wilder, Russell M.	Rochester
§	Williams, Henry L., Jr.	Rochester
§	Wilmer, Harry A.	Rochester
	Wilson, Paul F.	Rochester
§	Wilson, Robert B.	Rochester
§	Wilson, Viktor O.	Rochester
§	Winkelmann, Richard K.	Rochester
‡	Winter, Malcolm D., Jr.	Ellsworth A.F.B., South Dakota
†	Wise, James K.	Rochester
§	Wollaeger, Eric E.	Rochester
†§	Woltman, Henry W.	Rochester
*†§	Wood, Harry G.	Rochester
†	Woodington, George F.	Rochester
§	Woolner, Lewis B.	Rochester
†	Worlton, James T.	Rochester
	Worrell, Janet	Rochester
†	Wry, Paul E.	Rochester
§	Yoss, Robert E.	Rochester
§	Younger, Henry H.	Rochester
†	Zimmer, Roy W., Jr.	Rochester

Alphabetic Roster

Key to Symbols:
*Deceased; †Affiliate, Associate, Resident or Life Members; ‡In Service; ¶Honorary;
§Wife is Member of Woman's Auxiliary.

† Aanes, Almer M.	Red Wing
Aaro, Leonard A.	Rochester
Abraham, Arden L.	Duluth
Abrams, Alexander, Jr.	St. Paul
Abramson, Milton	Minneapolis
Achor, Richard W. P.	Rochester
Adair, Albert F., Jr.	St. Paul
† Adams, Bertram S.	Hibbing
Adams, Harold R.	St. Paul
† Adams, Ralph E.	Rochester
† Addison, Robert M.	Rochester
Addy, Edward R.	Gilbert
Adkins, Charles D.	Minneapolis
Adkins, Galen H.	Sandstone
Adkins, James T.	Bertha
Adson, Martin A.	Rochester
Affeldt, Daniel E.	Kasson
Aga, John H.	Brainerd
† Ager, Ernest A.	Minneapolis
Agustsson, Hreidar	Minneapolis
Ahern, Eugene E.	Minneapolis
Ahern, Gene J.	Red Wing
Ahlfs, Jacob J.	Caledonia
Ahlstrom, Robert C.	Braham
Ahola, Kenneth E.	Hibbing
† Ahrens, Albert E.	St. Paul
Ahrens, Curtis F.	Duluth
Ahrens, Robert M.	St. Paul
Akins, Willard M.	Red Wing
Albrecht, Harold H.	Lindstrom
Alcorn, William J.	Wabasso
Alden, John F., Jr.	St. Paul
Alden, W. Charles	Kimball
Alexander, Harlan A.	Minneapolis
Aling, Charles A.	Minneapolis
† Allaman, Loren E.	Rochester
Allen, Edgar V. N.	Rochester
Allen, John H.	Montevideo
Allison, David D.	Litchfield
Alpert, Abraham E.	Virginia
† Altnow, Hugo O.	Coral Gables, Fla.
Alton, Donald G.	Bird Island
Amatuzio, Donald S.	Minneapolis
† Amberg, Samuel	Rochester
Ambrus, Laszlo	St. Paul
Amerongen, W. W.	St. Paul
Andersen, Howard A.	Rochester
Andersen, Silas C.	Minneapolis
Anderson, Arden O.	Brainerd
Anderson, Arnold S.	St. Louis Park
Anderson, Chester A.	Hector
Anderson, Chester A.	Madison
Anderson, David M.	St. Louis Park
Anderson, David P., Jr.	Austin
† Anderson, Edward D.	Gstaad, Switzerland
Anderson, Edward M.	St. Cloud
Anderson, Ernest R.	Minneapolis
Anderson, Frank J.	Minneapolis
Anderson, Franklin C.	Owatonna
Anderson, Harold J.	Austin
Anderson, James J.	Mankato
Anderson, John A.	Minneapolis
Anderson, John T.	Minneapolis
Anderson, John W.	Blue Earth
Anderson, Karl W.	Minneapolis
Anderson, Margaret C.	Mankato
Anderson, Mark J.	Rochester
Anderson, Markham, J., Jr.	Rochester
Anderson, Milton W.	Rochester
Anderson, Richard E.	Willmar
Anderson, Richard W.	Aurora
Anderson, Roger L.	Minneapolis
Anderson, U. Schuyler	Minneapolis
Anderson, Waldo P.	Buffalo
Anderson, Wallace E.	Clearbrook
Anderson, Wallace E.	Minneapolis
Anderson, Wallace R.	Austin
Anderson, Werner W.	Brainerd
Anderson, William H.	Minneapolis
Anderson, William T.	Minneapolis
Andreassen, Einer C.	Minneapolis
Andreassen, Rolf L.	Minneapolis
Andresen, Karl d'A	Minneapolis
Andrew, William F.	Duluth
Andrews, Robert S.	Minneapolis
† Andrews, Roy N.	Mankato
Ankner, Frank J.	Minneapolis
Anonsen, Richard E.	Minneapolis
Ansell, Julian S.	Minneapolis
Antonow, Arthur M.	Virginia
† Anzel, Sanford H.	Rochester
† Arends, Archabald L.	Minneapolis
Arey, Stuart Lane	Minneapolis
Arhelger, Stuart W.	Minneapolis
Arlander, Clarence E.	Minneapolis
Arling, Leonard S.	Minneapolis
Arms, James J.	Minneapolis
Armstrong, Byron H.	Hopkins
Armstrong, Ralph S.	Winnebago
Arnesen, John F.	Owatonna
Arneson, Arthur I.	Morris
Arnold, Ann W.	Minneapolis
Arnold, Elmer Wm.	Adrian
† Arnold, John W.	Rochester
Arnquist, Andrew S.	St. Paul
Arny, Frederick P.	St. Paul
Arvidson, Carl G.	Minneapolis
Arzt, Philip K.	St. Paul
Asta, Joseph J.	Duluth
Athens, Alvin G.	Duluth
Atmore, William G.	Duluth
Aufderheide, Arthur C.	Duluth
Aulick, Ernest J.	Paynesville
*†Aune, Martin	Minneapolis
Aurelius, J. Richards	St. Paul
Ausman, Duane R.	St. Paul
Austrian, Sol	St. Paul
Autrey, William A.	St. Cloud
‡ Axler, Gueri	New York
Baars, Conrad W.	Rochester
Babb, Frank S.	St. Paul
Backus, Reno W.	Nopeming
Badeaux, George I.	Brainerd
Bader, J. L.	Slayton
Baer, Walter	St. Paul
Baggenstoss, Archie H.	Rochester
Baggenstoss, Osmond J.	Minneapolis
Bagley, Charles M.	Duluth
Bagley, Elizabeth C.	Duluth
Bagley, Russell W.	Minneapolis
Bagley, William R.	Duluth
Baich, Velemir M.	Coleraine
Bair, Hugo L.	Rochester
Baird, Joseph W.	Minneapolis
Baken, Melvin P.	Minneapolis
Baker, Abe B.	Minneapolis
† Baker, Alfred T.	Minneapolis
Baker, George S.	Rochester
† Baker, Harry R.	Hayfield
Baker, Hillier L., Jr.	Rochester
Baker, Jeannette L.	Fergus Falls
Baker, Milton E.	Minneapolis
Baker, Norman H.	Fergus Falls
Bakke, Arnold C.	Minneapolis
Bakkila, Henry E.	Duluth
Balcome, Milton M.	St. Paul
Baleisis, Peter	Minneapolis
† Balfour, Donald C.	Rochester
Balfour, William M.	Lawrence, Kansas
Balkin, Samuel G.	Minneapolis
Balmer, Albert I.	Duluth
Balogh, Charles J.	Minneapolis
Balz, George P.	Toledo, Ohio
Bank, Harry E.	Unknown
Banner, Edward A.	Rochester
Barber, Tracy E.	Austin
Bardon, Richard	Duluth
Bargen, J. Arnold	Rochester
Barker, John D.	Duluth
Barker, Nelson W.	Rochester
† Barnes, Arlie R.	Rochester
Barnes, Richard E.	Aurora
Barnett, Joseph M.	St. Paul
Barnett, Robert M.	Minneapolis
† Barney, Leon A.	Duluth
Barno, Alex	St. Louis Park
‡ Baronofsky, Ivan D.	New York
Barr, James S.	Elmore
Barr, Lowell C.	Albert Lea
Barr, Maxwell M.	Minneapolis
Barr, Robert N.	Minneapolis
Barr, Ronald W.	Montevideo
Barrett, Earl E.	Duluth
Barron, Jesse J.	Minneapolis
† Barron, Moses	Minneapolis
Barron, S. Steven	Minneapolis
Barry, Maurice J., Jr.	Rochester
† Barsness, Nellie O. N.	St. Paul
Bartholomew, Lloyd G.	Rochester
Bartness, John	Albert Lea
Bartzen, Peter J.	Duluth
Basinger, Harold P.	Windom
Basinger, Homer P.	Windom
Bastron, James A.	Rochester
Batdorf, B. Niles	Mankato
Bauer, Eugene L.	St. Paul
Bauer, Paul G.	Faribault
Baumgartner, Florian H.	Albany
Baylay, E. Covell	Lake City
Bayrd, Edwin D.	Rochester
Beach, Northrop	Minneapolis
Beahrs, Oliver H.	Rochester
† Beals, Hugh	LaJolla, Calif.
Bean, Charles N.	Waconia
† Beargie, Richard J.	Rochester
Beaton, J. Gordon	Northfield
Beck, Charles J.	No. St. Paul
† Becker, Arnetta M.	Lincoln, Neb.
Becker, Frederick T.	Duluth
Beckering, Gerrit	Edgerton
Bedford, Edgar Wm.	Minneapolis
Beech, Raymond H.	St. Paul
Beek, Harvey O.	St. Paul
Beer, John J.	St. Paul
Behmler, Frederick Wm.	Morris
Behr, Orlo K.	Crookston
Beirstein, Samuel	Minneapolis
Beiswanger, Richard H.	Minneapolis
Belcher, R. A.	Little Falls
Bell, Charles C.	St. Paul
† Bell, Elexious T.	Minneapolis
Bellomo, James	St. Paul
† Bellomo, John	St. Paul
Bellville, Titus P.	Minneapolis
Belzer, Meyer S.	Minneapolis
Bender, James H.	Brainerd
Bendix, Lester H.	Annandale
† Benedict, William L.	Rochester
Benell, Otto E.	Virginia
Benepe, James L.	St. Paul
Benesh, Louis A.	Minneapolis
Benjamin, Edwin G.	Minneapolis
Benjamin, Harold G.	Minneapolis
Benjamin, Walter G.	Pipestone
Benson, Alfred H.	Little Falls
Benthack, Elaine M.	St. Paul
Bepko, Marie K.	Cloquet
Berdez, George L.	Duluth
Berg, Arnold M.	Roseau
Berg, Clinton C.	Wayzata
† Bergan, Otto	Clinton
Bergan, Robert O.	Duluth
Berge, David O.	Roseau
Berge, Harry L.	Mora
Berge, Kenneth G.	Rochester
Berger, Alex G.	Minneapolis
Bergh, George S.	Minneapolis
Bergh, Solveig M.	Minneapolis
Berglund, Eldon B.	Minneapolis
Bergman, Oscar B.	St. James
Bergquist, James R.	Minneapolis
*†Berkman, David M.	Oronoco
Berkman, John M.	Rochester
Berkwitz, Nathaniel J.	Minneapolis
† Berman, Harold J.	Rochester
† Berman, Irwin B.	Rochester
Berman, Reuben	Minneapolis
† Berman, Stanley	Rochester
Bernatz, Philip E.	Rochester
Bernhisel, Harris G.	Minneapolis
Bernier, M. J.	No. St. Paul
Bernstein, Irving C.	Minneapolis
Bernstein, William C.	St. Paul
Berris, Harold	Minneapolis
Bessesen, Alfred N., Jr.	Minneapolis
Beuning, John B.	St. Cloud
Bevis, William D.	Minneapolis
Bianco, Anthony J.	Duluth
Bicek, Joseph F.	St. Paul
Bickel, William H.	Rochester
† Biedermann, Jacob	Thief River Falls
Bieter, Raymond N.	Minneapolis
† Bigelow, Charles E.	Dodge Center

† Bigg, Richard L.	Rochester
Bigler, Earl E.	Perham
Bigler, Ivan E.	Perham
Bilka, Paul J.	Minneapolis
Billings, Harry H.	Red Wing
Billings, Ralph E.	Franklin
† Billingsley, John S.	Rochester
Binder, Manuel R.	Minneapolis
† Binet, Henry E.	Grand Rapids
† Binger, Henry E.	Phoeniz, Ariz.
Birkhead, Newton C.	Rochester
Bissinger, Lester L.	Brainerd
Bjornson, Robert G. B.	Minneapolis
Black, B. Marden	Rochester
Black, Earl J.	St. Paul
Black, William A.	New Ulm
Blackburn, Charles M.	Rochester
Blackburn, Henry W.	St. Paul
Blackmore, Sidney C.	Biwabik
Blake, Allan J.	Hopkins
Blake, James A., Jr.	Hopkins
Blake, Paul S.	Hopkins
† Blakey, Adam R.	Osakis
Blakey, Leslie W.	Rochester
† Bledsoe, Francis H.	Rochester
Bloedel, Traugott J.	Osseo
Blomberg, Robert D.	Minneapolis
Blomberg, William R.	St. Paul
Bloom, Joseph	Silver Bay
Bloom, Norman B.	Minneapolis
Blumberg, Henry B.	St. Paul
Blumenthal, Jacob S.	Minneapolis
Boardman, Dalmon V.	Winona
† Bock, Rolland A.	St. Paul
Bodaski, Albert A.	Tyler
Boehrer, John J., Jr.	Minneapolis
Bofenkamp, Benjamin	Minneapolis
Bofenkamp, F. William	Luverne
Bohn, Donald G.	Minneapolis
Boies, Lawrence R.	Minneapolis
* Bolender, Harold L.	St. Paul
Boline, Clifford A.	Battle Lake
† Bolsta, Charles	Ortonville
Bolstad, Owen C.	Little Falls
Bolz, J. Arnold	Grand Rapids
Boman, Paul G.	Duluth
Bonello, Frank J.	St. Paul
Bonner, John L.	Eveleth
Bonnet, John D.	Rochester
Boody, George J., Jr.	Sandstone
† Boolukos, George P. N.	Rochester
Boone, Ervin S.	Luverne
† Booth, Albert E.	Minneapolis
† Boreen, Clifton A.	Minneapolis
Borg, Joseph F.	St. Paul
Borgerson, Arthur H.	Long Prairie
† Borgeson, Egbert J.	St. Paul
Borman, Chauncey N.	Minneapolis
Boroson, Hugh N.	Thief River Falls
Borowicz, Leonard A.	Minneapolis
Bosland, Howard G.	Willmar
† Bossert, Clarence S.	Mora
‡ Boswell, J. Thornton	Kenosha, Wisc.
Bottolfson, Bottolf T.	Moorhead
Bouchelle, McLemore	Virginia
† Bouma, Lewis R.	St. Paul
Bouquet, Bertram J.	Wabasha
Bouthilet, Florence J.	St. Paul
† Bowen, Ralph, Jr.	Rochester
Bowen, Robert L.	Hibbing
Bowers, Gordon G.	Minneapolis
Bowers, Robert N.	Lake City
Boyd, David A., Jr.	Rochester
Boyd, Frank E.	Jasper
Boyd, George K.	Rochester
Boyer, George S.	Crookston
Boyer, Samuel H., Jr.	Duluth
Boyle, Francis J.	Tracy
Boynton, Ruth E.	Minneapolis
Boysen, Herbert	Madelia
† Braasch, William F.	Rochester
Brabec, Paul F.	Hastings
Bradley, Jeannne B.	Minneapolis
Bradley, John G.	Minneapolis
Bradley, Nelson J.	Willmar
Brand, George D.	St. Paul
† Brand, William A.	Redwood Falls
Brandenburg, Robert O.	Rochester
Brandt, Henry E.	Minneapolis
† Branham, Donald S.	Deer Park, Wisc.
Brannick, Thomas L.	Rochester
Bratholdt, James W.	Watertown
† Bratrud, Arthur F.	Minneapolis
† Bratrud, Edward	Thief River Falls
Bratrude, Earl J.	St. James
Braun, Ohrmundt C.	Grand Rapids
Bravick, Donald D.	Minneapolis
† Bray, Elwyn R.	St. Paul
Bray, Philip N.	Duluth
Breitenbucher, Robert B.	Minneapolis
Brekke, Harvey J.	Minneapolis
Brennan, Joseph G.	Browerville
Bretzke, Carl O.	Hutchinson
Bridge, Allyn G.	Minneapolis
Briggs, John F.	St. Paul
Brigham, Charles F., Jr.	St. Cloud
Brill, Alice K.	Minneapolis
Brink, Adlai A.	Baudette
Brink, Donald M.	Hutchinson
Broadbent, James C.	Rochester
Broadie, Thomas E.	St. Paul
Brockway, Roger W.	Grand Rapids
Brodie, Walter D.	St. Paul
Broker, Henry M.	St. Cloud
† Brooker, Warren J.	Minneapolis
† Brooks, Charles N.	Minneapolis
Brow, Raymond E.	Rochester
Brown, Alexander E.	Rochester
Brown, Cyrus C., Jr.	Duluth
† Brown, Edgar D.	Unknown
Brown, Henry A.	Rochester
Brown, Ian A.	St. Paul
‡ Brown, James E., Jr.	St. Paul
Brown, Joe R.	Rochester
† Brown, John C.	Los Gatos, Calif.
Brown, Philip W.	Rochester
Brown, William D.	Minneapolis
Bruhl, Heinz H.	Faribault
Brunsting, Louis A.	Rochester
Brusegard, James F.	Red Wing
Bucher, Foster D.	Starbuck
Buchstein, Harold F.	Minneapolis
† Buck, Frederick H.	Shakopee
Buckley, J. J.	Edina
Buckley, Robert P.	Duluth
Buesgens, Ralph H.	Waterville
† Buie, Louis A.	Rochester
Buirge, Raymond E.	Minneapolis
Bulinski, Theodore J.	St. Paul
† Bulkley, Kenneth	Minneapolis
Burch, Edward P., II	St. Paul
*† Burch, Frank E.	St. Paul
Burchell, Howard B.	Rochester
‡ Buresh, Kenneth L.	Unknown
Burger, Thomas C.	Evansville, Indiana
Burgert, E. O., Jr.	Rochester
Burich, Harry F.	Rochester
Burke, Edmund C.	Rochester
Burklund, Edwin C.	St. Paul
† Burleigh, Edward G., Jr.	Minneapolis
Burlingame, David A.	St. Paul
Burman, Richard E.	Aitkin
Burmeister, Richard O.	St. Paul
Burnett, Joseph W.	New Ulm
Burnham, Wesley H.	Minneapolis
† Burns, Catherine	Albert Lea
Burns, Floyd M.	Milan
Burns, M. Alpheus	Milan
† Burns, Robert M.	St. Paul
Burseth, Edgar C.	Mora
Burton, Carl G.	St. Paul
Buscher, Julius C.	St. Cloud
Bush, Robert P.	St. Paul
Bushard, Wilfred J.	Minneapolis
Busher, Herbert H.	St. Paul
Butler, John K.	Cloquet
Butt, Hugh R.	Rochester
Butturff, Carl R.	Freeborn
† Butzer, John A.	Mankato
Butzer, John F.	Mankato
Buzzelle, Leonard K.	Minneapolis
Byrne, William J.	St. Paul
Cable, Morris L.	Minneapolis
Cabot, Clyde M.	Minneapolis
† Cadman, Norman L.	Rochester
Cady, Laurence H.	Minneapolis
Cain, Clark L.	St. Paul
Cain, James C.	Rochester
Cain, James H.	Alexandria
Cairns, Robert J.	Redwood Falls
† Calhoun, Frank W.	Albert Lea
Calin, Stanford H.	St. Paul
Callahan, Francis F.	St. Paul
Callahan, John A.	Rochester
Callan, Joseph A.	Virginia
‡ Callewart, Robert A.	Texas
† Calverley, John R.	Rochester
Cameron, Dale C.	St. Paul
Cameron, Isabell L.	Minneapolis
Cameron, John H.	Crookston
Camp, Ray Junior	Madison
Campbell, Dennis V.	Fergus Falls
Campbell, Donald C.	Rochester
† Campbell, Lowell M.	Minneapolis
Campbell, Malcolm K.	Rochester
Campbell, Orwood J.	Minneapolis
Canfield, Wayne W.	St. Peter
Canine, James L.	So. St. Paul
Cantwell, William F.	International Falls
Caplan, Leslie	Minneapolis
Card, William H.	Minneapolis
Cardle, George E.	Brainerd
Carey, James B.	Minneapolis
Carey, James B., Jr.	Minneapolis
Carlander, Lester W., Jr.	Minneapolis
Carley, Walter A.	St. Paul
Carlon, Donald L.	Willmar
Carlson, Carl E.	Alexandria
Carlson, Charles V.	Mound
Carlson, Homer J.	Pelican Rapids
Carlson, John V.	Westbrook
Carlson, Lawrence	Minneapolis
Carlson, Leonard T.	Minneapolis
‡ Carlson, Robert G.	Seattle, Wash.
Carlson, Russell E.	Stillwater
Carlson, Vernon J.	Moorhead
† Carnevali, John F.	Rochester
Caron, Robert P.	Minneapolis
Carr, David T.	Rochester
Carr, William J.	Minneapolis
Carroll, John J.	Winsted
Carryer, Haddon McC.	Rochester
Carthey, Frank J.	New Ulm
Caspers, Carl G.	Minneapolis
† Catlin, John J.	Buffalo
Catlin, Theodore J.	Buffalo
Cauble, Charles F.	Minneapolis
† Cavanor, Frank T.	Minneapolis
Cawein, Madison	Rochester
Cedarleaf, Cherry B.	Mahtomedi
Ceder, Elmer T.	Minneapolis
Cella, Joseph A.	Minneapolis
Ceplecha, Stanley F.	Redwood Falls
Cervenka, Charles F.	New Prague
Cesnik, Robert J.	Sauk Rapids
Chadbourn, Charles R.	St. Paul
Chadbourn, Wayne A.	Minneapolis
Chalgren, William S.	Mankato
Challman, S. Alan	Minneapolis
† Chambers, Winslow C.	Blue Earth
Chappell, Elliott R.	Stillwater
† Chatterton, Carl C.	St. Paul
† Chavez, Demetrio A.	St. Paul
Chermak, Francis G.	International Falls
Chervenak, William A.	Winsted
Chesler, Merrill D.	Minneapolis
Child, Sherman B.	Minneapolis
Childs, Donald S., Jr.	Rochester
Chisholm, Tague C.	Minneapolis
Chittum, John R.	Aurora
Christensen, Clarence H.	Duluth
Christensen, Cli E.	Winona
Christensen, Llewellyn E.	Minneapolis
Christensen, Norman A.	Rochester
Christiansen, Andrew	St. Paul
Christiansen, Harold A.	Jackson
Chunn, Stanley S.	Willmar
Ciriacy, Edward W.	Ely
Clagett, O. Theron	Rochester
Clapp, Hubert D.	Crookston
Clark, Clarence L.	Duluth
Clark, Edward C.	Rochester
Clark, Elizabeth A.	Duluth
Clark, Harry B.	St. Cloud
Clark, Henry B., Jr.	Minneapolis
Clark, Ivan T.	Duluth
Clark, Leslie W.	Spring Valley
Clark, Malcolm D.	Minneapolis
Clark, Robert S.	Minneapolis
Clarke, John W.	Waconia
Clay, Lyman B.	Minneapolis
Cleaves, William D.	Sauk Centre
*† Clement, John B.	Lester Prairie
Clifford, George W.	Alexandria
† Clifton, Theodore A.	Hollywood, Fla.
Climie, Andrew R.	Rochester
Closuit, Frederick C.	Aitkin
‡ Clowdus, Bernard F.	Bolling A.F.B., D. C.
Cochrane, Byron B.	St. Paul
Cochrane, Ray F.	Minneapolis
Coddon, Walter D.	St. Paul
Coe, John I.	Minneapolis
† Coffey, William F. X.	Rochester
Cohen, Bernard A.	Minneapolis
Cohen, Ellis N.	Minneapolis
Cohen, Ephriam B.	Minneapolis
Cohen, Henry W.	Minneapolis
† Cohen, Maynard M.	Minneapolis
Cohen, Sumner S.	Oak Terrace
Colby, Malcolm Y., Jr.	Rochester
Colby, Woodard L.	St. Paul
Cole, James S.	Minneapolis
Cole, Wallace H.	St. Paul
Coleman, John B.	St. Paul
Coll, James J.	Duluth
† Collie, Henry G.	St. Petersburg, Fla.
† Collins, Arthur N.	Duluth
† Collins, Joseph S.	Wabasha
*† Combacker, Leon C.	Fergus Falls
* Comfort, Mandred W.	Rochester
Conley, Francis W.	Duluth
Conley, Robert H.	Mankato
Connolly, Coleman J.	St. Paul
Connolly, Daniel C.	Rochester
Connolly, Joseph P.	So. St. Paul
Connor, Charles E.	St. Paul

† Connor, P. James .... Rochester
Cook, C. Kenneth .... St. Paul
Cook, Edward N. .... Rochester
Cook, Jay M. .... Staples
Coombs, Carl H. .... Cass Lake
Cooper, Charles C. .... St. Paul
Cooper, John P. .... Minneapolis
† Cooper, Maurice D. .... Winnebago
Cooper, Robert R. .... Minneapolis
Cooper, Talbert .... Rochester
Cope, Hershel B. .... Virginia
Corbin, Kendall B. .... Rochester
Corniea, Albert D. .... Minneapolis
† Corr, William P. .... Rochester
Correa, Dale H. .... Minneapolis
Corrigan, Cyril J. .... Rochester
‡ Coseriu, Vasile G. .... St. Paul
Cosgriff, James A. .... Olivia
Cosgriff, James A., Jr. .... Olivia
‡ Cottone, Francis J. ..Trenton, New Jersey
Coulter, Harold E. .... Madelia
Courteau, Robert D. .... Onamia
Courtright, Claiborne .... Rochester
* Covell, Walter W. .... St. Peter
Coventry, Markham B. .... Rochester
Coventry, William D. .... Duluth
Covey, Kenneth W. .... Rochester
Cowan, Donald W. .... Minneapolis
Cowan, George M. .... Duluth
Craig, Clair C. .... International Falls
Craig, David M. .... St. Paul
Craig, M. Elizabeth .... Minneapolis
† Craig, Winchell McK. .... Rochester
† Cranmer, Richard R. .... Minneapolis
Cranston, Robert W. .... Minneapolis
Creevy, Charles D. .... Minneapolis
Cronwell, Bernhard J. .... Austin
Crowley, James H. .... St. Paul
Crudo, Vincent D. .... St. Paul
Crump, James W. .... St. Paul
* Culligan, John M. .... St. Paul
Culligan, Leo C. .... Minneapolis
Culp, Ormond S. .... Rochester
Culver, L. G. .... St. Paul
Cumming, E. Dale .... St. Paul
Cundy, Donald T. .... Minneapolis
Curtis, Rauen A. .... LeCenter
Cushing, Richard T. .... St. Louis Park
Cushing, Robert L. .... Brainerd
† Cutts, George .... Minneapolis

*†Dady, Elmer E. .... Minneapolis
Daehlin, Rolf .... Fergus Falls
Daggett, Donald R. .... Minneapolis
Dahl, Elmer O. .... Minneapolis
Dahl, James C. .... Minneapolis
Dahl, John A. .... Minneapolis
Dahlin, David C. .... Rochester
† Daignault, Oscar .... Benson
Dale, Lester N. .... Red Lake Falls
Daly, David D. .... Rochester
Danford, Harold G. .... Appleton, Wisc.
† Daniel, Donald H. .... Minneapolis
† Danielson, Karl A. .... Litchfield
Danielson, Lennox .... Litchfield
Danyluk, Michael .... Minneapolis
Dargay, Cyril P. .... Minneapolis
Daugherty, Guy W. .... Rochester
David, Reuben .... Hopkins
† Davidson, Paul .... Rochester
Davidson, William D. .... St. Joseph
Davis, Arthur E., Jr. .... St. Cloud
† Davis, Austin C. .... Florida
Davis, Edward V. .... St. Paul
Davis, George D. .... Rochester
Davis, Jay C. .... Minneapolis
Davis, Lloyd T. .... Wadena
Davis, Luther F. .... Wadena
Davis, Raymond D. .... Waseca
Davis, William I. .... Sandstone
Dawson, James R. .... St. Paul
Dawson, Lorin D. .... Worthington
Dearing, William H. .... Rochester
Decker, Barry .... Rochester
Decker, Charles H. .... St. Paul
Decker, David G. .... Rochester
Degallier, Daniel .... Winona
DeKruif, Hendrik .... Fergus Falls
*†Delmore, John L. .... Roseau
Delmore, John L., Jr. .... Roseau
† Delmore, Robert J. ....Inglewood, Calif.
del Plaine, Carlos W. .... Minneapolis
Demo, Robert A. .... Albert Lea
Derauf, Benjamin I. .... St. Paul
Derauf, Donald E. .... St. Paul
Deters, Donald C. .... St. Paul
Detjen, Edward D. .... Bigfork
Deutsch, Robert J. .... Duluth

Devereaux, Thomas J. .... Wayzata
Devine, Kenneth D. .... Rochester
† DeWall, Richard A. .... Minneapolis
DeWeerd, James H. .... Rochester
Deweese, Wilford J. .... Bemidji
Dewey, Donald H. .... Owatonna
Dickman, Roy W. .... Minneapolis
Dickson, Franklin H., Jr. .... Proctor
Diefenbach, Eugene J., Jr. ....Minneapolis
Diehl, Harold S. .... New York
Dierker, Heinrich A. .... Minneapolis
Diessner, Ardell W. .... Redwood Falls
Diessner, Grant R. .... Rochester
Diessner, Henry D. .... Minneapolis
Dille, Donald E. .... Litchfield
Dillenburg, C. J. .... Little Falls
Divertie, Matthew .... Rochester
Dixon, Claude F. .... Rochester
Doan, Robert E. .... Wayzata
† Dobbs, Richard L. .... Rochester
Dobler, Manfred D. .... Ely
Dobson, Mervin W. .... Mankato
Dockerty, Malcolm B. .... Rochester
Docksey, John W. .... Willmar
Dodds, William C. .... Detroit Lakes
Dodge, Henry W., Jr. .... Rochester
Dodson, Albertus F. .... Brainerd
Doherty, Elmer M. .... New Prague
Dokken, James H. .... Windom
Doman, Victor Wm. .... Lakefield
† Dombrowski, Edmund T. .... Rochester
Doms, Vernon A. .... Elbow Lake
Donaldson, Charles S. .... St. Cloud
Donatelle, Edward P. .... Minneapolis
Donoghue, Francis E. .... Rochester
Donovan, Daniel L. .... Albert Lea
Dordal, John .... Sacred Heart
Dorge, Richard I. .... Minneapolis
Dorsey, George C. .... Minneapolis
Doscherholmen, Alfred .... Minneapolis
Douglass, Bruce E. .... Rochester
‡ Dovenmuehle, Robert H...Durham, N. C.
† Doxey, Gilbert L. .... Minneapolis
† Doxsee, George C. ....Iowa City, Iowa
Doyle, James R. .... Rochester
Doyle, Lawrence O. .... Minneapolis
Drake, Carl B. .... St. Paul
† Drake, Charles R. .... Minneapolis
† Dredge, Homer P. .... Minneapolis
Drexler, George W. .... Blue Earth
Drill, Herman E. .... Hopkins
† Drips, Della G. .... Oronoco
Dry, Thomas J. .... Cape Town, Africa
† DuBois, Julian F. .... Sauk Centre
DuBois, Julian F., Jr. .... Sauk Centre
Duff, Edwin R. .... Minneapolis
Dummer, Donald J. .... New Brighton
Duncan, James W. .... Moorhead
† Dungay, Neil S. .... Northfield
Dunitz, Norman L. .... Rochester
Dunlap, Earl H. .... Minneapolis
* Dunn, James N. .... St. Paul
Dunn, Robert C. .... St. Paul
Dupont, Joseph A. .... Excelsior
Duryea, Marbry .... Minneapolis
Duryea, Willis M. .... Minneapolis
Duryea, Willis M., Jr. .... Minneapolis
DuShane, James W. .... Rochester
Dvorak, Benjamin A. .... Minneapolis
Dwan, Paul F. .... Minneapolis
Dworsky, Samuel D. .... Minneapolis
Dwyer, John J. .... Duluth
Dyer, John Allen .... Rochester
Dyrdal, Paul J. .... St. Paul
Dysterheft, Adolf F. .... Gaylord

Earl, George A. .... St. Paul
Earl, John T. .... St. Paul
Eaton, Lealdes M. .... Rochester
Eberley, Tobe S. .... Benson
† Eberlin, Edward A. .... Glenwood
Eckdale, John E. .... Marshall
Eckman, Philip F. .... Duluth
Eckman, Ralph J. .... Duluth
Edelmann, Robert B.....Short Hills, N. J.
† Edelstein, Jack P. .... Rochester
Eder, Walter P. .... Minneapolis
Edwards, Jesse E. .... Rochester
Edwards, Joseph W. .... St. Paul
Edwards, Lloyd G. .... St. Paul
Edwards, Thomas J. .... St. Paul
Edwin, Russell L. .... Rochester
Egge, Sanford G. .... Albert Lea
Eginton, Charles T. .... St. Paul
Ehrenberg, Claude J. .... Minneapolis
Ehrlich, S. Paul .... Minneapolis
Eich, Matthew A. .... Minneapolis
Eichhorn, Edmund P., Jr. .... Minneapolis
Eide, O. A. .... Hancock

Eiler, John .... Park Rapids
Eisenman, Walter .... Hibbing
Eisenstadt, David H. .... Minneapolis
Eisenstadt, William S. .... Minneapolis
Eitel, George D. .... Minneapolis
† Ekblad, John W. .... Rock Island, Ill.
Eklund, Carl D. .... Duluth
Ekstrand, LeRoy M. .... Wabasha
Eli, Earl W. .... Minneapolis
Elias, Frank J. .... Duluth
Elkins, Earl C. .... Rochester
Ellertson, Leonard M. .... Albert Lea
Ellinger, Albert J. .... Willmar
Elliott, Harold J. .... Hayfield
Ellis, Earl Wm. .... Elgin
Ellis, F. Henry .... Rochester
Ellison, David E. .... Minneapolis
Ellison, Ellis .... Minneapolis
Ellison, Evan S. .... Minneapolis
Ellison, Frank E. .... Monticello
Ellwood, Paul M., Jr. .... Minneapolis
Elrod, Calvin R. .... Long Lake
† Elsey, James R. .... Glenwood
* Ely, Orriman S. .... So. St. Paul
Emanuel, Karl W. .... Duluth
Emerson, Edward C. .... St. Paul
Emerson, Edwin E. .... Osakis
Emmett, John L. .... Rochester
Emmons, R. W. .... St. Paul
Emond, Albert J. .... Farmington
Emond, Joseph S. .... Farmington
Endress, Edward K. .... St. Paul
Engberg, Edward J. .... Faribault
Engel, Joseph P. .... Minneapolis
Engelhart, Peter C. .... Minneapolis
† Engler, Robert S. .... Rochester
Englund, Elvin F. .... Minneapolis
Englund, Garth W. .... Mabel
Engstrand, Oscar J. .... Minneapolis
Engstrom, Denton P. .... Minneapolis
Engstrom, Robert B. .... Mankato
Enroth, Oscar E. .... St. Paul
Eppard, Raymond M. .... Cloquet
Erdal, Ove A. .... Albert Lea
Erich, John B. .... Rochester
Erickson, Alvin O. .... Long Prairie
Erickson, Clifford O. .... Minneapolis
Erickson, Donald J. .... Rochester
Erickson, Eskil .... Halstad
Erickson, George P. .... Hibbing
Erickson, Laurence F. .... Minneapolis
Erickson, Myron E. .... Minneapolis
Erickson, Reuben F. .... Minneapolis
Erickson, Vernon D. .... Grand Rapids
† Ericson, Swan .... Los. Angeles, Calif.
Erlanson, Andrew C. .... St. Paul
† Ernest, George C. H. ....
St. Petersburg, Fla.
Ersfeld, Murray P. .... St. Paul
† Erskine, Gordon M. .... Grand Rapids
*†Ertel, Edward Q. .... Ellendale
Esensten, Sidney .... Minneapolis
† Eshelby, E. C. .... St. Paul
‡ Esser, Robert A. .... Paris, France
Estes, J. Earle .... Rochester
Estrem, Ralph L. .... Fergus Falls
Estrem, Robert D. .... Fergus Falls
† Eusterman, George B. .... Rochester
Eustermann, John J. .... Mankato
Evans, Edward T. .... Minneapolis
Evans, Harold W.....Grand Forks, N. D.
Evans, Leslie M. .... Sauk Rapids
Evans, Robert D. .... Minneapolis
† Evanson, David J. .... Rochester
† Evarts, Arrah B. .... Rochester
Evensta, John B. .... Grand Rapids
Ewen, Edgar F. .... Rochester
Ewens, George B. .... Virginia

Faber, John E. .... Rochester
† Fahr, George E. .... Minneapolis
Failor, Harlan J. .... Rochester
Fallon, Virgil T. .... Dawson
Falls, John L. .... Red Wing
Fansler, Walter A. .... Minneapolis
Farkas, John V. .... St. Paul
Fasbender, Herman T. .... Hastings
Faucett, Robert L. .... Rochester
Faulconer, Albert, Jr. .... Rochester
Fawcett, Arthur M. .... Renville
Fawcett, Keith R. .... Duluth
Fedor, Robert D. .... Litchfield
Fee, John G. .... St. Paul
Feeney, John M. .... Minneapolis
Feigal, David W. .... Wayzata
Feigal, William M. .... Thief River Falls
Feinberg, Milton .... Sacramento, Calif.
Feinberg, Philip .... Minneapolis

Feinberg, Samuel B..........Minneapolis
Feinberg, Walter D..........Rochester
‡ Feinerman, Burton..........New York
Feinstein, Julius Y..........Minneapolis
Felder, Davitt A..........St. Paul
Feldmann, Floyd M......New York, N. Y.
Felion, Arthur J..........St. Paul
Fellows, Manley F..........Duluth
Fenger, Ejvind P. K..........Oak Terrace
Ferguson, William C..........Walnut Grove
Ferrand, Paula T..........Moose Lake
Ferrell, Clarence R..........Grand Rapids
Ferris, Deward O..........Rochester
Fesenmaier, Otto B..........New Ulm
Fesler, Harold H..........St. Paul
Fetzek, Albert D..........Austin
Feuling, John C..........Duluth
Fidelman, Norman E..........Foley
Field, Anthony H..........Farmington
Field, Charles W..........Minneapolis
Fifer, William R..........Minneapolis
Fifield, Malcolm M..........Duluth
† Figi, Frederick A..........Rochester
Fingerman, David L..........Minneapolis
Fink, Daniel L..........St. Paul
Fink, Leo W..........Minneapolis
Fink, Robert J..........Minneapolis
Fink, Walter H..........Minneapolis
Finkelnburg, William O..........Winona
Fisch, Herbert M..........Austin
Fischer, John R..........Blooming Prairie
Fischer, Mario McC..........Duluth
Fischer, Robert F..........St. Paul
Fisher, Dan W..........St. Paul
Fisher, Isadore I..........Minneapolis
Fisketti, Henry..........Duluth
† Fitzgerald, Don F..........Wayzata
Fitzsimons, William E., Jr..........Brainerd
† Fjeldstad, C. Alford..........Minneapolis
Flanagan, Harold F..........St. Paul
Flanagan, Leonard G..........Austin
Flancher, Leon H..........Crookston
Flannery, Hubert F..........St. Paul
Flatt, John R..........Wabasha
Fleeson, William H..........Minneapolis
Fleming, Dean S..........Hopkins
† Fleming, Thomas N..........St. Cloud
Flesche, Bernard A..........Lake City
Fliehr, Richard R..........Minneapolis
Flink, Edmund B..........Minneapolis
Flinn, James B..........Redwood Falls
† Flinn, Thomas E..........Remer
Floersch, Adrian J..........Owatonna
Flom, Reynold P..........St. Paul
† Flom, Robert S..........Minneapolis
Florine, Martin C..........Janesville
Flynn, Bernard F..........Hibbing
Flynn, L. L., Jr..........St. Paul
† Fogarty, Charles W..........St. Paul
Fogarty, Charles W., Jr..........St. Paul
Fogelberg, Emil J..........St. Paul
Foker, Leslie W..........Minneapolis
Foley, Frederic E. B..........St. Paul
Folken, Frank G..........Albert Lea
Folsom, Louis B..........Minneapolis
Ford, Burton C..........Marshall
Ford, F. Wendell..........New London
Ford, William H..........Minneapolis
Forsythe, James R..........St. Paul
Fortier, George M. A..........Little Falls
Fortier, Rene G..........Mankato
Foss, Edward L..........Rochester
Foster, Orley W..........Minneapolis
Foulk, William T., Jr..........Rochester
† Fowler, L. Haynes..........Minneapolis
† Fox, Donald P.....Tanganyika, East Africa
Fox, James Rogers..........Minneapolis
Fox, LeRoy J..........St. Paul
† Franchere. Frederick Wm.......Lake Crystal
Francis, David W..........Morristown
† Franckowiak, John J..........Rochester
Frane, Donald B..........Minneapolis
Fredericks, George M..........Minneapolis
Frederickson, Alice C..........Willmar
* Fredricks, Merriam G..........Duluth
Freeman, Charles D., Jr..........St. Paul
‡ Freeman, Craig W..........Minneapolis
Freeman, Donald W..........St. Louis Park
Freeman, Gerald I..........St. Paul
† Freeman, John P..........Glenville
Freidman, Louis L..........St. Paul
French, Bayard T..........Hibbing
French, Lyle A..........Minneapolis
Frethem, Allen A..........Rochester
Frey, Richard J..........Minneapolis
Friberg, Joseph B..........Minneapolis
Fricke, Robert E..........Rochester
Fried, Louis A..........Minneapolis
Friedell, Aaron..........Minneapolis
Friedell, George..........St. Louis Park
Friedlieb, Oskar P..........Minneapolis
Friedman, Harry S..........Minneapolis
Friend, Charles A..........Minneapolis
Fritsche, Albert..........New Ulm
Fritsche, Carl J..........New Ulm
Fritsche, Theodore R..........New Ulm
Fritz, Wallace L..........St. Paul
Froats, Charles W..........St. Paul
Frost, John B..........Minneapolis
Frost, Russell H..........Oak Terrace
Frykman, Howard M..........Minneapolis
Fryling, Vera B..........Agnew, Calif.
Frys, Russell N..........Minneapolis
*†Fugina, George R..........Mankato
Fuller, Alice H..........Minneapolis
Fuller, Benjamin F..........St. Paul
Fuller, Josiah..........Duluth
Funk, Victor K..........Oak Terrace
Furlow, William L..........Faribault
Furman, Lucie C..........Minneapolis
† Furr, Leo O..........Unknown
Fusaro, Ramon M..........Minneapolis

Gaard, Richard C..........Minneapolis
Gaebe, Milton B..........Clara City
Gaida, Joseph B..........St. Cloud
Gailitis, Veronika M..........Cambridge
† Gallagher, Bernard J..........Waseca
Gallett, Lester E..........Minneapolis
Galligan, John J..........St. Paul
† Galligan, Margaret M.D..........Minneapolis
† Gambill, Carl M..........Rochester
Gambill, Earl E..........Rochester
Gamble, Elbert J..........Albert Lea
Gamm, Edgar R..........Triumph
Gammell, John H..........Minneapolis
Garamella, Joseph J..........Minneapolis
Garbrecht, Arthur Wm..........St. Paul
Gardner, Gawinn B..........Rochester
Gardner, Jack K..........Ceylon
Gardner, John U..........Rochester
Gardner, Victor H..........Fairmont
Gardner, Walter P..........St. Paul
Garlock, DeWitt H.......Highlands, Calif.
Garrow, Douglas M..........St. Paul
Garske, George L..........Minneapolis
Garten, Joseph L..........Minneapolis
Garvey, James T..........Minneapolis
Gastineau, Clifford F..........Rochester
† Gatzke, Laurence D..........Rochester
Gault, N. L., Jr..........Minneapolis
Gault, Sarah J..........Minneapolis
Gaviser, David..........Minneapolis
Gehlen, Joseph N..........St. Paul
Geib, Marvin J..........Fargo. No. Dak.
Geiser, Peter M..........Alexandria
George, Vane P., Jr..........Minneapolis
Geraci, Joseph E..........Rochester
Geurs, Benjamin R..........Mankato
Ghormley, Ralph K..........Carmel, Calif.
† Ghostley, Mary C..........Santa Ana, Calif.
Gibbs, Edward C..........St. Paul
Gibbs, Robert W..........Minneapolis
Giebenhain, John N..........Minneapolis
Giere, Joseph C..........Minneapolis
Giere, Richard W..........Minneapolis
Giere, Silas W..........Benson
Giesbrecht, A. ....Hallock
† Giffin, Herbert Z..........Rochester
Giffin, Mary E..........Rochester
Gifford, R. W., Jr..........Rochester
Gilbert, Maurice G..........Minneapolis
Gildersleeve, Robert..........Watertown
† Giles, William F..........Rochester
‡ Gill, Charles R..........Ft. Meade, Md.
Gill, Theodore..........Albert Lea
Gillespie, Delmar R..........St. Paul
Gillespie, Malcolm G..........Duluth
† Gilloon, James R..........Rochester
Gilman, Lloyd C..........Willmar
*†Gilmore, Rowland..........Puposky
Gilpin, Charles A., Jr..........Rochester
Gilsdorf, Donald A..........St. Paul
Gingold, Benjamin A..........Minneapolis
† Girvin, Richard B..........Des Peres, Mo.
Gislason, Paul H..........Mankato
Gislason, Solveig T..........St. Peter
Gjerde, William P..........Lake City
Glabe, Robert A..........Plainview
Glaeser, John H..........Minneapolis
Glassberg, Irving J..........Minneapolis
Glathe, John P..........Rochester
Gleason, Wallace A..........St. Paul
‡ Glick, Dallas D.........San Francisco, Calif.
Glueck, Bernard C., Jr..........Minneapolis
Goblirsch, Andrew P..........Sleepy Eye
Goehrs, Gilman H..........St. Cloud
† Goehrs, Henry W..........St. Cloud
Goetz, Frederick C..........Minneajolis
Gold, David..........Minneapolis
Goldberg, Isadore M..........Minneapolis
Goldberg, Marvin E..........Minneapolis
Goldish, Daniel R..........Duluth
Goldish, Robert J..........Duluth
Goldman, Theodore I..........Minneapolis
Goldner, Meyer Z..........Minneapolis
Goldschmidt, Volker..........Duluth
Goldsmith, Joseph W..........St. Paul
Goldstein, Norman P..........Rochester
† Goltz, Edward V..........St. Paul
Goltz, Robert W..........Minneapolis
Good, C. Allen, Jr..........Rochester
Good, Hoff D..........Minneapolis
Good, Roy H..........Glenwood
Goodchild, William R..........Minneapolis
Goodman, Charles E..........Virginia
Goodnow, William H..........Duluth
Gordon, John R..........Minneapolis
Gordon, Philip E..........Minneapolis
Gordon, Sewell S..........St. Louis Park
‡ Gottlieb, Cornelius M.........Tacoma, Wash.
Gould, Allan B., Jr..........Rochester
Gowan, Lawrence R..........Duluth
† Graham, Archibald W..........Chisholm
Grahek, Jack P..........Ely
† Granquist, Richard D..........Minneapolis
Grant, Hendrie W..........St. Paul
Grant, J. C..........Sauk Centre
Grant, Suzanne..........Minneapolis
Grasse, John M., Jr.
Aibonito, Puerto Rico
† Gratzek, Frank R..........Minneapolis
† Gratzek, Thomas..........St. Paul
Grau, R. K..........St. Paul
† Graudins, Gunars..........Rochester
Graves, Richard B..........Red Wing
Gray, Edward F..........White Bear Lake
† Gray, Frank D..........Marshall
Gray, Royal C..........Minneapolis
Green, Clayton Robbie..........Excelsior
Green, Paul A..........Rochester
Green, Robert A..........St. Louis Park
Greenberg, Albert J..........Minneapolis
Greene, Daniel E..........Thief River Falls
Greene, Laurence F..........Rochester
Greenfield, Irving..........Minneapolis
Greenfield, William T..........Cokato
Greenspan, Richard H..........Minneapolis
† Greishiemer, Esther M.....Philadelphia, Pa.
Gridley, John W..........Arlington
Griebie, Grant L..........Brownton
Griffin, John W., Jr..........Bemidji
Griffin, Richard P..........Benson
Grimes, Burton P..........St. Peter
Grimes, Marian..........Minneapolis
Grimes, Paul T..........Park Rapids
Grimmell, Francis J..........Minneapolis
Grindlay, John H..........Rochester
Grinley, Andrew V..........Grand Rapids
Grise, William B..........Austin
Grizzle, Claude O..........Rochester
Grohs, William H..........Duluth
Gronvall, Paul R..........Minneapolis
Groschupf, Richard P..........Bemidji
Groschupf, Theodore P..........Bemidji
Grose, Frederick N. ....Clarissa
Gross, John B..........Rochester
Grotting, John K..........Minneapolis
* Grundset, Ole J..........Montrose
* Guilbert, G. D..........Minneapolis
Guilfoile, Pierre J..........Delano
Guldseth, Gustav J..........Hendricks
† Gullickson, Andrew..........Minneapolis
Gullickson, Glenn, Jr..........Minneapolis
Gully, Raymond J...Fort Meade, So. Dak.
Gunlaugson, Frederick G..........Minneapolis
Gushurst, Edward G..........
Palm Springs, Calif.
Gustafson, Paul O..........Minneapolis
Gustason, Harold T..........Minneapolis
Guy, Jack A..........New London
Gyde, Maurice C..........Rochester

Haas, Jack F..........Northfield
Haase, Donald D..........Silver Bay
Haavik, John E..........Duluth
Habein, Harold C..........Rochester
Habein, Harold C., Jr..........Unknown
Haberer, Helen R..........Minneapolis
‡ Haberland, Lyle F..........New York, N. Y.
Haberle, Charles A..........Minneapolis
† Haberman, Emil..........Osakis
Haes, Julius E..........Mankato
Haesly, Warren W..........Winona
Hagedorn, Albert B..........Rochester
Hagen, John D..........Austin
Hagen, Kristofer..........Minneapolis
† Hagen, Olaf J..........Moorhead
Hagen, Wayne S..........Minneapolis
Hogen, Paul S..........St. Paul
† Haggard, George D..........Minneapolis
† Haines, Samuel F..........Rochester
Hakanson, Erick Y..........St. Paul
Halbert, John J..........Duluth
Halenbeck, Philip L..........St. Cloud

Hall, Barnard	St. Paul
Hall, Harry B.	Minneapolis
Hall, Wendell H.	Minneapolis
Hall, William E.	Maple Lake
Hallberg, Olav E.	Rochester
† Hallenbeck, Dorr F.	LaJolla, Calif.
Hallenbeck, George A.	Rochester
Halliday, Phillip V.	Duluth
Hallin, Roger P.	Worthington
Halloran, Walter H.	Jackson
Halme, William B.	Wadena
Halper, Bernard	Hibbing
Halpern, David J.	Brewster
Halpin, Joseph E.	Rush City
Halverson, Donald E.	Winnebago
Halverson, Kermit J.	Chisholm
Halverson, William G.	Madelia
Halvorsen, Daniel K.	Owatonna
Halvorson, James W.	Zumbrota
Hamel, Joseph I.	Minneapolis
Hamilton, Samuel L.	Red Wing
‡ Hamilton, Warren W.	Rochester
Hamlon, John S.	Fergus Falls
Hammer, Lawrence M.	Mankato
Hammerstrom, Robert N.	Minneapolis
Hammes, Ernest M.	St. Paul
Hammes, Ernest M., Jr.	St. Paul
† Haney, Claude L.	Duluth
Hankerson, Robert G.	Minnesota Lake
Hanlon, David G.	Rochester
Hannah, Hewitt B.	Minneapolis
Hannon, Donald W.	St. Paul
Hanover, Ralph D.	International Falls
Hansen, Cyrus O.	Minneapolis
Hansen, Erling W.	Minneapolis
Hansen, Milo L.	Little Falls
Hansen, Olga S.	Minneapolis
Hansen, Robert E.	Hibbing
Hansen, Rollin M.	Minneapolis
Hansen, Theodore M.	Albert Lea
Hanske, Edward A.	Minneapolis
† Hanson, Adolph M.	Faribault
Hanson, Ernest O.	Cloquet
Hanson, Everett C.	New York Mills
Hanson, Harlow J.	Minneapolis
Hanson, Harold B.	Minneapolis
† Hanson, Henry V.	St. Paul
Hanson, John W.	Northfield
† Hanson, LeRoy W.	Bellaire, Texas
Hanson, Lewis	Frost
Hanson, Malcolm B.	Minneapolis
Hanson, Mark C. L.	Minneapolis
Hanson, Norbert O.	Rochester
Hanson, William A.	Minneapolis
Happe, Lawrence J.	Minneapolis
Harada, Thomas T.	Lake Wilson
Harbaugh, John T.	Neosho, Mo.
Hargraves, Malcolm M.	Rochester
Harmon, Gaius E.	St. Paul
Harrington, Stuart W.	Rochester
Harrington, Vernon A., Jr.	Duluth
Harris, Leon D.	Minneapolis
Harris, Lloyd E.	Rochester
Harrison, Edgar G., Jr.	Rochester
Harrison, Percy W.	Worthington
Harrison, William C.	Minneapolis
Hart, William E.	Monticello
Hartfiel, William F.	St. Paul
Hartig, Marjorie	St. Paul
Hartjen, Jason K.	Bemidji
Hartman, Evelyn E.	Minneapolis
† Hartman, Howard R.	Rochester
Hartnagel, Grant F.	Red Wing
Hartridge, Virginia B.	Rochester
Hartung, Elmer H.	Claremont
Hartwich, Roger F.	Winona
Hass, Frederick M.	Minneapolis
Hassel, Carl W., Jr.	Rochester
† Hassett, Roger G.	Mankato
Hastings, DeForest R.	Minneapolis
Hastings, Donald W.	Minneapolis
† Hatch, Walter E.	Duluth
Hauge, Erling T.	Minneapolis
Hauge, Malvin I.	Clarkfield
Haugen, George W.	Minneapolis
Haugen, John A.	Minneapolis
Hauser, Donald C.	Minneapolis
Hauser, George W.	Minneapolis
† Hauser, Harris M.	Rochester
Hauser, Victor P.	St. Paul
Havel, Robert J.	Minneapolis
Haven, Walter K.	Minneapolis
† Havens, Fred Z.	Riverside, Calif.
† Havens, John G. W.	Austin
Hawk, Dale J.	St. Charles
Hawkinson, Raymond P.	Minneapolis
Hawley, George M. B., II	Red Wing
Hay, Lyle J.	Minneapolis
Hayes, Albert F.	St. Paul
† Hayes, James M.	Arcadia, Calif.
Hayles, Alvin B.	Rochester
Hays, Albert T.	Minneapolis
† Haywa, Eugene W.	Minneapolis
Head, Douglas P.	Minneapolis
† Healy, Raymond T.	Pierz
Hebbel, Robert	Minneapolis
† Hebeisen, Milton B.	Carver
Heck, Frank J.	Rochester
Heck, William W.	St. Paul
Hedemark, Homer H.	Ortonville
Hedemark, Truman A.	Ortonville
Hedenstrom, Frank G.	St. Paul
Hedenstrom, Paul H.	Cambridge
Hedenstrom, Philip C.	Marshall
Hedlund, Charles J.	St. Paul
Hedrick, William L.	Rochester
Heegaard, William G.	Alexandria
† Hegge, Olav H.	Austin
Heiam, William C.	Cook
† Heiberg, Emmett A.	Fergus Falls
Heiberg, Olaf M.	Worthington
Heid, James K.	Little Falls
Heilig, William R.	St. Paul
† Heilman, Dorothy M. H.	Long Beach, Calif.
Heilman, Fordyce R.	Rochester
Heimark, John J.	Mankato
Heimark, Julius J.	Fairmont
Heinz, Ivy B.	Shakopee
Heinz, John N.	Wadena
Heinz, Lawrence H.	Shakopee
Heinzerling, Carl R.	Chaska
Heise, Carl vR.	Winona
Heise, Paul vR.	Winona
Heise, Philip vR.	Winona
† Helland, John W.	Unknown
Heller, Edgar E.	Mankato
† Helmholz, Henry F.	Rochester
Helseth, Hovald K.	Fergus Falls
Helwig, Karl L., Jr.	Kerkhoven
† Hempel, Dean J.	Rochester
† Hempstead, Bert E.	Rochester
Henderson, Arthur J. G.	No. St. Paul
Henderson, Edward D.	Rochester
Henderson, John W.	Rochester
Henderson, Lowell L.	Rochester
Hendricks, Esten J.	Verndale
† Hendrickson, John F.	Minneapolis
Hengstler, William H.	St. Paul
Hendrikson, Earl C.	Minneapolis
Henry, Clarence J.	Milaca
† Henry, Clifford E.	Kirksville, Mo.
Henry, Harold W.	Hinckley
Henry, Joseph E.	Milaca
Henry, Kenneth G.	Owatonna
Henry, Martin R.	St. Peter
† Hensel, Charles N.	St. Paul
Hepper, Norman G.	Rochester
Herbert, Willis L.	Minneapolis
Herbst, Richard F.	Willmar
Herman, Samuel M.	St. Paul
Hermann, Harold W.	Minneapolis
Hermanson, Peter E.	Hendricks
Herrmann, Edgar T.	St. Paul
† Hertel, Garfield E.	Austin
Hertz, Myron J.	St. Paul
Hesla, Inman A.	Austin
† Hewitt, Edith S.	Topeka, Kansas
† Hewitt, Richard M.	Topeka, Kansas
*†Heyerdale, Oscar C.	Rochester
Higgins, Dennis C.	Rochester
Higgins, John A.	Rochester
† Higgins, John H.	Minneapolis
Hildebrand, John E.	Bemidji
Hilding, Anderson C.	Duluth
*†Hilger, Andrew Wm.	St. Paul
Hilger, Jerome A.	St. Paul
Hilger, Laurence D.	St. Paul
Hilgermann, George O.	Minneapolis
Hilker, Marcus D.	St. Paul
Hill, Earl	Minneapolis
Hill, Elmer M.	Minneapolis
† Hill, Frederick E.	Riverside, Calif.
† Hill, John P.	Lima, Ohio
Hill, John R.	Rochester
Hill, Richard Woolsey	Rochester
† Hillis, Samuel J.	E. Bradenton, Fla.
Himebaugh, Gilbert J.	Evansville, Ind.
Hinckley, Robert G.	Minneapolis
Hinderaker, Harris P.	Bird Island
Hines, Edgar A., Jr.	Rochester
Hiniker, Louis P.	St. Paul
Hiniker, Peter J.	Le Sueur
Hinz, Walter E.	Willmar
† Hirschboeck, Frank J.	Duluth
Hirsh, Stanton A.	Crookston
Hirshfield, Frank R.	Minneapolis
Hitchcock, Claude R.	Minneapolis
Hochfilzer, John J.	St. Paul
Hodapp, Robert V.	Willmar
Hodges, Kenneth V.	Minneapolis
Hodgson, Corrin H.	Rochester
Hodgson, Jane E.	St. Paul
Hodgson, John R.	Rochester
Hoeg, Dwight C.	Duluth
Hoeper, Philip G.	Mankato
Hoff, Herbert O.	Duluth
Hoffbauer, Frederick W.	Minneapolis
Hoffert, Henry E.	Minneapolis
Hoffman, Roy A.	Minneapolis
Hoffman, Walter L.	Minneapolis
Hoganson, Donald E.	Bemidji
† Hoidale, Andrew D.	Tracy
† Holcomb, Joel T.	Marine-on-St. Croix
† Holcomb, O. Wm.	St. Paul
Holian, Darwin K.	Albert Lea
Holland, C. R.	Rochester
Hollenhorst, Robert W.	Rochester
Hollinshead, W. H.	St. Paul
Holm, Donald F.	Benson
Holman, Colin B.	Rochester
Holmberg, Conrad J.	Minneapolis
† Holmberg, LeRoy J.	Canby
Holmen, Robert W.	St. Paul
Holmes, Alva E.	Rush City
Holmstrom, Carle H.	Warren
Holt, John E.	St. Paul
Holzapfel, Fred C.	Minneapolis
Hom, Leong Y. W.	Battle Lake
Honath, Donald H.	Owatonna
† Hooker, John P.	Rochester
† Hoover, Norman W.	Rochester
† Hopkins, Donald M.	Rochester
Hopkins, G. Wendell	St. Paul
Hoppes, Emerson E.	Minneapolis
Horns, Howard L.	Minneapolis
Horns, Richard C.	Minneapolis
† Horton, Bayard T.	Rochester
Hottinger, Raymond C.	Janesville
Houglum, Arvid J.	Lake Park
Houkom, Bjarne	Minneapolis
Houkom, Samuel S.	Duluth
Houle, Rollin J.	New Brighton
* Houston, Donald M.	Park Rapids
Houts, Joseph C.	Dassel
Hovde, Rolf	Winthrop
Hovland, Melvin L.	Minneapolis
Howard, Frank M., Jr.	Rochester
Howard, Marshall I.	Mankato
Howard, Merrill A.	St. Paul
Howard, Robert B.	Minneapolis
Howard, Solomon E.	Minneapolis
Howe, Newell W.	St. Paul
Howell, Carter W.	Minneapolis
Howell, Llewelyn P.	Rochester
Howell, Milton M.	Glencoe
Howland, Willard J., Jr.	Rochester
Hoyer, Ludolf J.	Windom
† Hoyt, Charles S.	Rochester
Hruza, William J.	Madelia
Hubin, Edwin G.	Sandstone
Hudec, Elwyn R.	Echo
Hudson, George E.	Minneapolis
Huebert, Dan W.	Hutchinson
† Huenekens, Edgar J.	Minneapolis
† Huff, James F.	Rochester
Huff, J. S.	Owatonna
† Huffington, Herbert L.	Lutsen
Huffington, Herb L., Jr.	Waterville
Hughes, Bernard J.	Brainerd
Hughes, Sidney O.	Winona
Huizenga, Kenneth A.	Rochester
Hullsiek, Harold E.	St. Paul
* Hullsiek, Richard B.	Fort Snelling
Hulteng, Donald B.	Minneapolis
Hultkrans, Rudolph E.	Minneapolis
Humphrey, Edward W.	Moorhead
† Humphrey, Wade R.	Stillwater
Hunt, Arthur B.	Rochester
† Hunt, James C.	Rochester
Hunt, James Calvin	Rochester
† Hunt, Roscoe C.	Fairmont
Hunt, William	Fergus Falls
Hunter, James S., Jr.	Rochester
Hunter, Murray H.	Farmington
Hunter, Samuel W.	St. Paul
† Hurd, Annah	Minneapolis
Hurwitz, Milton M.	St. Paul
Husebye, Kjeld O.	St. Paul
Hustad, Edward G.	Montevideo
Hutchinson, Dorothy W.	Oak Terrace
Hutchinson, Henry	Moose Lake
† Huxley, Frederick R.	Fairbault
Hymes, Charles	Minneapolis
† Hynes, John E.	Minneapolis

† Ide, Arthur W.	White Bear Lake
Ide, Arthur W., Jr.	Minneapolis
Idstrom, Linneus G.	Minneapolis
Ikeda, Kano	St. Paul
Indeck, Walter	Minneapolis
† Indihar, John E., Jr.	Minneapolis
Ingalls, Edgar G., Jr.	Minneapolis
Inglis, William	Redwood Falls
Ireland, Gerald W.	Grand Rapids
† Irvine, Harry G.	Minneapolis
Iverson, Rolf M.	Minneapolis
Ivins, John C.	Rochester
Ivy, Horace K.	Rochester

Jackman, Raymond J. ....Rochester
Jackson, William C. ....St. Paul
Jacobs, Douglas L. ....Willmar
† Jacobs, Johannes C. ....Willmar
Jacobson, Clarence ....Chisholm
Jacobson, Clifford W. ....Breckenridge
Jacobson, Ferdinand C. ....Duluth
Jacobson, Loren J. ....Minneapolis
Jacobson, Wyman E. ....St. Louis Park
Jahnke, William R. ....St. Paul
James, Ellery M. ....St. Paul
James, John W. ....Mahtomedi
Janecky, Allen G. ....Baudette
Janes, Joseph M. ....Rochester
Janssen, Martin E. ....St. Paul
† Jarvis, Bruce W. ....St. Paul
Jarvis, Charles W. ....St. Paul
Jarvis, Marilyn A. ....St. Paul
Jastram, Rupert M. ....St. Paul
Jay, Alan R. ....Minneapolis
Jefferies, William L. ....St. Louis Park
Jensen, Alvin M. ....Brownton
Jensen, Harry C. ....Minneapolis
Jensen, John A. ....Crookston
† Jensen, Marius J. ....Minneapolis
Jensen, Nathan K. ....Minneapolis
Jensen, Reynold A. ....Minneapolis
† Jensen, Thorvold J. ....Duluth
Jenson, James E. ....Stillwater
Jepson, William W. ....Minneapolis
Jerome, Bourne ....Minneapolis
Jerome, Elizabeth K. ....Minneapolis
Jeronimus, Henry J. ....Duluth
† Jesion, Joseph W. ....Pine River
Jessico, Charles M. ....Duluth
Jeub, Robert P. ....Minneapolis
Jewell, A. H. ....Austin
Joffe, Harold H. ....Virginia
Johanson, Waldemar G. ....St. Paul
John, Robert H. ....Duluth
Johnsen, Henry A., Jr. ....Edina
Johnson, Adelaide McF. ....Rochester
Johnson, Aldridge F. ....Isle
Johnson, Angelo G. ....Minneapolis
Johnson, Arthur B. ....Minneapolis
Johnson, Calvin J. ....Grand Rapids
Johnson, Carl E. ....St. Paul
Johnson, Carl E. ....Rochester
Johnson, Carolyn A. ....St. Paul
Johnson, C. Percy ....Tyler
Johnson, Curtis M. ....Winona
Johnson, David R. ....Minneapolis
Johnson, Douglas L. ....Little Falls
Johnson, Edward A. ....Minneapolis
† Johnson, Einer W. ....Bemidji
Johnson, Einer, W., Jr. ....Rochester
Johnson, Frank E. ....Minneapolis
Johnson, Harry A. ....Minneapolis
Johnson, Herbert W. ....St. Paul
* Johnson, Hobart C. ....No. Mankato
† Johnson, James A. ....Minneapolis
Johnson, John W. ....Minneapolis
*† Johnson, Julius ....Minneapolis
Johnson, Karl E. ....Duluth
Johnson, Malcolm R. ....Minneapolis
Johnson, Marvin W. ....Dassel
Johnson, Morris B. ....Minneapolis
Johnson, Norman P. ....Minneapolis
Johnson, Norman Paul ....Minneapolis
Johnson, Norton T. ....Minneapolis
Johnson, Olga H. ....Moorhead
Johnson, Orville H. ....Edina
Johnson, Paul E. ....Minneapolis
Johnson, R. J. ....St. Paul
Johnson, Ralph B. ....Lanesboro
‡ Johnson, Ray A. ....New York, N. Y.
Johnson, Reinald G. ....Minneapolis
Johnson, Reuben A. ....Minneapolis
Johnson, Richard S. ....Minneapolis
Johnson, Robert E. ....Minneapolis
Johnson, Robert H. ....Chisago City
Johnson, Roger S. ....Wayzata
Johnson, Victor ....Rochester
Johnson, Vilhelm M. ....Dawson
Johnson, William E. ....Morgan
Johnson, Youbert T. ....Minneapolis
Johnsrud, Luverne W. ....Hibbing
Johnston, Henry W. ....Virginia
Johnston, Leonard F. ....Winona
† Jolin, Francis M. ....Grand Rapids
*† Jones, Alvah W. ....Red Wing
Jones, David G. ....Minneapolis
† Jones, E. Mendelssohn ....St. Paul
§ Jones, Herbert W., Jr. ....Minneapolis
Jones, Orville H. ....Mankato
Jones, Richard H. ....Minneapolis
† Jones, Richard N. ....St. Cloud
Jones, William R. ....Minneapolis
† Jordan, Kathleen B. Smith....Granite Falls
Jordan, Lewis S. ....Granite Falls
Jorgenson, Edward O. ....Duluth
Josewich, Alexander ....Minneapolis
Josewski, Raymond J. ....Stillwater
Joyce, George L. ....Rochester
Judd, Allen S. ....Minneapolis
Judd, Edward S., Jr. ....Rochester
Judd, Walter H. ....Washington, D. C.
Juergens, Herman M. ....Belle Plaine
Juergens, John L. ....Rochester
Juergens, Manley F. ....Stillwater
Juers, Edward H. ....Red Wing
† Juliar, Richard O. ....Los Angeles, Calif.
Juntunen, Roy R. ....Duluth
Jurdy, Mitchell J. ....Minneapolis
Just, Herman J. ....Hastings

† Kaasa, Lawrence J. ....Albert Lea
† Kabrick, Ola A. ....St. Peter
Kadesky, Harold B. ....Minneapolis
Kaiser, Milton L. ....New Ulm
Kalin, Oscar T. ....Minneapolis
Kallestad, Leonard L. ....Wayzata
Kaman, Gordon R. ....St. Paul
Kanne, Earl R. ....Brainerd
Kantar, Bruce L. ....Minneapolis
Kaplan, David H. ....St. Paul
Kaplan, Harold A. ....Minneapolis
Kaplan, J. Jacob ....Minneapolis
Kapsner, Alfred T. ....Princeton
Karansky, Stanley ....Phoenix, Ariz.
Karges, Laurel E. ....Grand Rapids
Karleen, Bernard N. ....Jackson
Karleen, Conrad I. ....Minneapolis
Karlen, Markle ....Minneapolis
Karn, Jacob F. ....Ortonville
Karon, Irvine M. ....St. Paul
Kasper, Eugene M. ....St. Paul
Kasper, Robert E. ....Minneapolis
Kath, Reinhard H. ....Arlington
Katz, Louis J. ....Long Beach, Calif.
Katz, Yale J. ....Los Angeles, Calif.
Kaufman, Edward J. ....Appleton
Kaufman, Herschel J. ....Minneapolis
Kaufman, Walter B. ....Mankato
Kaufman, William C. ....Appleton
Kavanaugh, Gerald J. ....Rochester
Kazdan, Martin S. ....Rochester
Kearney, Rochfort W. ....Mankato
Kearns, Thomas P. ....Rochester
Keating, Francis R., Jr. ....Rochester
Keefe, Rolland E. ....St. Paul
Keil, Marcus A. ....Albert Lea
Keith, Haddow M. ....Rochester
† Keith, Norman M. ....Rochester
Keith, Paul J. ....Milaca
Keithahn, Elmer E. ....Sleepy Eye
Kelby, Gjert M. ....Minneapolis
Kelley, Roger E. ....Crosby
Kelley, Walter M. ....Duluth
Kelly, Albert C. ....St. Paul
Kelly, Charles F. ....Minneapolis
Kelly, Edward H. ....St. Paul
Kelly, James H. ....St. Paul
Kelly, John F. ....Cold Spring
Kelly, John P. ....Minneapolis
*† Kelly, John V. ....St. Paul
Kelly, Patrick J. ....Rochester
Kelly, Robert T. ....Grand Rapids
† Kelsey, Carleton G. ....St. Paul
Kelsey, Chauncey M. ....St. Paul
† Kemp, Alphonse F. ....Mankato
Kenefick, Emmett V. ....St. Paul
Kennedy, Byrl J. ....Minneapolis
† Kennedy, Claude C. ....Minneapolis
Kennedy, George L. ....Faribault
† Kennedy, Jane F. ....Minneapolis
Kennedy, Roger L. J. ....Rochester
Kenyon, Thomas J. ....St. Paul
Kerkhof, Arthur C. ....Minneapolis
Kernohan, James W. ....Rochester
Kesting, Herman ....St. Paul
Kevern, Jay L. ....Henning
Keyes, John D. ....Winona
Keyes, Robert W. ....Pipestone
Kiely, Joseph M. ....Rochester
Kierland, Robert R. ....Rochester
Kiesler, Frank, Jr. ....Minneapolis
Kilbride, Edwin A. ....Worthington
Kim, Mark K. ....St. Cloud
Kimmel, George C., Jr. ....Minneapolis
Kincaid, Owings W. ....Rochester
King, Frances W. ....Oak Terrace
† King, George L. ....Hudson, Wisc.
† King, Harold E. ....Rochester
Kinkade, Byron R. ....Ada
Kinports, Edward B. ....International Falls
Kinsella, Thomas J. ....Minneapolis
† Kinzel, Raymond C. ....Rochester
Kiolbasa, Edward B. ....Stillwater
Kippen, Neil ....Breckenridge
Kirby, Thomas J., Jr. ....Rochester
Kirklin, John W. ....Rochester
† Kirshen, Robert ....Rochester
*† Kistler, Alvin J. ....Minneapolis
Kitzberger, Peter J. ....New Ulm
Kjenaas, Ervin A. ....Hastings
Klass, Donald W. ....Rochester
Klefstad, Lloyd H. ....Greenbush
† Klein, Harry ....Duluth
Klein, William A. ....Duluth
*† Klima, William W. ....Stewart
† Kline, Richard F. ....St. Cloud
Knapp, Frank N. ....Duluth
Knapp, Miland E. ....Minneapolis
† Knight, Ralph T. ....Minneapolis
† Knight, Ray R. ....Minneapolis
Knoche, Harvey A. ....Morgan
Knoedler, John P. ....Duluth
Knoll, W. V. ....Brainerd
Knudsen, Helen L. ....Minneapolis
Knutson, Gerhard E. ....St. Paul
Knutson, Kenneth R. ....Hibbing
Knutson, Lewis A. ....Spring Grove
Knutson, Robert C. ....St. Paul
Kodres, Nina ....St. Paul
Koelsche, Giles A. ....Rochester
Koenecke, F. H. ....Lakefield
† Koenig, Robert P. ....Minneapolis
† Koenigsberger, Charles ....Mankato
Koepcke, Gerald M. ....Minneapolis
Kohlbry, Carl O. ....Duluth
† Kohler, Delphin W. ....Tacoma, Wash.
Kohlhase, Robert E. ....Minneapolis
Kolars, James J. ....Faribault
Koller, Hermann M. ....Minneapolis
Koller, Louis R. ....Minneapolis
Koller, Robert L. ....Hopkins
Kolp, Berton A. ....Glenwood
Kooda, Jennings C. ....Morris
Koons, W. R. ....Mahnomen
Koop, Herman E. ....Cold Spring
‡ Koop, Severin H., Jr. ....Sauk Rapids
Korchik, John P. ....Minneapolis
Korda, Henry A. ....Pelican Rapids
Kosiak, John, Jr. ....Minneapolis
Kosiak, William ....Two Harbors
Kostick, William R. ....Fertile
Kotchevar, Frank R. ....Eveleth
Kottke, Frederic J. ....Minneapolis
Kotval, Russell J. ....Pipestone
Koucky, Rudolph W. ....Minneapolis
‡ Kovack, Freeman D. ....Amarillo, Texas
Koza, Donald W. ....St. Paul
Kozberg, Oscar ....Moose Lake
Kraemer, George N. ....Fairmont
Krafft, Walter E. ....Minneapolis
Kragh, Lyle V. ....Rochester
Krause, Carl W. ....Fairmont
Kremen, Arnold J. ....Minneapolis
Kreuzer, Titus C. ....Marshall
Krezowski, Thomas K. ....St. Paul
Krieser, Albert E. ....Minneapolis
Krueger, Victor R. ....Nopeming
Kruger, Elmer L., Jr. ....Nashwauk
Krusen, Frank H. ....Rochester
Kruzich, Stephen J. ....Sleepy Eye
† Krystosek, Lee A. ....Madison, Wisc.
Kucera, Frank J. ....Hopkins
Kucera, Stanley T. ....Northfield
† Kucera, William J. ....
Santa Barbara, Calif.
Kugler, Alex A. ....St. Paul
Kuhlmann, Lawrence B. ....Melrose
Kulstad, Oscar S. ....Dodge Center
Kulzer, Norbert J. ....Hastings
Kump, Warren L. ....Minneapolis
‡ Kurtin, Joseph J. ....New York, N. Y.
Kuske, Albert W. ....St. Paul
† Kusske, Arthur L. ....New Ulm
Kusske, Bradley W. ....St. Paul
Kusske, Douglas R. ....St. Paul
Kusz, Clarence V. ....Minneapolis
Kvale, Walter F. ....Rochester
* Kvitrud, Gilbert ....St. Paul

LaBree, John W. ....St. Louis Park
LaBree, Robert H. ....Duluth
LaFond, Edward M. ....St. Cloud
Lagaard, Sheldon M. ....Minneapolis
Laikola, Leslie A. ....Adrian
† Laird, Arthur T. ....Duluth
† Lajoie, John M. ....Minneapolis
Lake, Clifford F. ....Rochester
Lamb, H. Douglas ....Minneapolis
Lang, Leonard A. ....Minneapolis
Lannin, Bernard G. ....St. Paul
Lannin, Donald R. ....St. Paul
† Lansche, Richard K. ....Rochester
† Lapierre, Arthur P. ....Minneapolis
Larrabee, Walter F., Jr. ....St. Paul
Larsen, Frank Wm. ....Minneapolis

Larson, Arnold	Detroit Lakes
Larson, Arthur K.	Minneapolis
Larson, Arthur N.	Minneapolis
Larson, Clarence M.	Minneapolis
Larson, Donald M.	Minneapolis
† Larson, Donald M.	Minneapolis
Larson, Dorette W.	Minneapolis
Larson, Eva-Jane O.	St. Paul
Larson, Gerald E.	Cambridge
Larson, James T.	So. St. Paul
Larson, Keith D.	Moose Lake
Larson, Kenneth R.	St. Paul
Larson, Lawrence M.	Minneapolis
Larson, Leighton W.	Waconia
Larson, Leonard M.	Oak Terrace
Larson, Leroy J.	Bagley
Larson, Martin L.	St. Paul
Larson, Milo H.	Lake Benton
† Larson, Norman E.	Rochester
Larson, Oliver E. H.	Zumbrota
Larson, Paul N.	Minneapolis
† Larson, Robert L.	Rochester
Larson, Roger C.	Minneapolis
Larson, Wyllis G.	Northfield
Laszewski, Franz von Zelberschwecht	St. Paul
Latterell, Kenneth E.	Duluth
Latts, Elliot M.	Minneapolis
† LaVake, Rae T.	Minneapolis
Law, Harrison E.	Virginia
Laymon, Carl W.	Minneapolis
Lazarte, Jorge A.	Rochester
† Leahy, Bartholomew	St. Paul
* Leavenworth, Richard O.	St. Paul
Leavenworth, Richard O., Jr.	St. Louis Park
Leavitt, Clark T.	Rochester
Leck, Paul C.	Austin
*†Leddy, Eugene T.	Rochester
Lee, Gordon E.	Glenwood
Lee, Hubert W.	Brainerd
Lee, Norman J.	Tracy
Lee, Robert E.	Rochester
Leek, Joseph H.	Duluth
Leemhuis, Andrew J.	Minneapolis
Lehoux, Louis M.	Quebec, Canada
Leibold, Herbert H.	Parkers Prairie
Leick, Richard M.	St. Paul
Leiferman, Robert J.	Minneapolis
† Leigh, John E.	Rochester
Leinonen, Wendla E.	Anoka
Leitch, Archibald	St. Paul
Leland, Harold R.	Minneapolis
Lenander, Mellvin E.	St. Peter
† Lenarz, Albert J.	Browerville
Lende, Norman	Faribault
Lenz, Joseph R.	Morton
Lenz, O. A.	Minneapolis
Leonard, Lawrence J.	Minneapolis
Leonard, Samuel	Minneapolis
† Leopard, Brand A.	Brownsville, Texas
Lepak, Francis J.	Duluth
Lepak, John A.	St. Paul
Leppo, N. Erkki A.	Duluth
† Lerche, William	Cable, Wisc.
Lerner, A. Ross	Minneapolis
L'Esperance, Bernard F.	Two Harbors
Lester, Malcolm J., Jr.	Truman
Lester, Richard G.	Minneapolis
Letson, Robert D.	Glenwood
Leven, N. Logan	St. Paul
Leverenz, Carleton W.	St. Paul
Levitt, George X.	St. Paul
Lewis, Arthur J.	Henning
Lewis, Charles W.	Henning
*†Lewis, Claude B.	St. Cloud
† Lewis, David R.	Rochester
Lewis, Joyce S., Jr.	Minneapolis
*†Lexa, Frank J.	Lonsdale
Lick, Charles L.	St. Paul
Lick, Louis C.	St. Paul
Lick, William J., Jr.	St. Paul
† Liedloff, Adolph G.	Mankato
Lien, Richard J.	St. Paul
Lienke, Roger I.	Minneapolis
Liffrig, William W.	Red Wing
† Lighbourn, Edgar L.	Hastings
Lilleberg, Norbert J.	St. Paul
Lillehei, C. Walton	Minneapolis
Lillehei, Elmer J.	Robbinsdale
*†Lillie, Harold I.	Rochester
Lillie, John C.	Rochester
Lima, Ludvig, R., Jr.	Montevideo
Limbeck, Donald A.	Le Sueur
‡ Lind, Carl J., Jr.	Fort McPherson, Ga.
Lindahl, Merlyn J.	Sherburn
Lindberg, Alfred L.	Wheaton
Lindberg, Arthur N.	Minneapolis
† Lindberg, Arvid C.	Minneapolis
Lindberg, Vernon L.	Minneapolis
Lindberg, Winston R.	Minneapolis
Lindblom, Alton E.	North Mankato
Lindblom, Maurice L.	Minneapolis
† Linde, Herman	Cyrus
Lindell, Robert	St. Paul
Lindeman, Raymond J.	Paynesville
Lindemann, Charles E.	Minneapolis
Linderholm, Bruce E.	Minneapolis
Lindgren, Russell C.	Minneapolis
Lindquist, Richard H.	Minneapolis
Lindquist, Richard H.	Rochester
Linke, Charles A.	Duluth
Linke, Clara L.	Duluth
Linner, Gunnar	Minneapolis
† Linner, Henry P.	Minneapolis
Linner, John H.	Minneapolis
Linner, Paul W.	Minneapolis
† Lipinski, Stanley W.	Memphis, Tenn.
Lippman, Emanuel S.	Minneapolis
Lippman, Hyman S.	St. Paul
Lippmann, Elmer W.	Hutchinson
Lipschultz, Oscar	Minneapolis
Lipscomb, Paul R.	Rochester
† Litchfield, John T.	Minneapolis
Litin, Edward M.	Rochester
Litin, Robert B.	Rochester
Litkewitsch, Helene	St. Paul
Litman, Abraham B.	Minneapolis
Litman, Samuel N.	Duluth
Litzow, Thaddeus J.	Rochester
Lober, Paul H.	Minneapolis
Loes, Louis A.	St. Cloud
Lofgren, Karl A.	Rochester
Lofsness, Stanley V.	St. Paul
Lofstrom, Dennis E.	Pine River
*†Logan, Archibald H.	Rochester
Logan, George B.	Rochester
† Logefeil, Rudolph C.	Minneapolis
Logothetis, John A.	Minneapolis
Lohmann, John G.	Pipestone
Loken, Selmer M.	St. Paul
Loken, Theodore	Ada
Lommen, Peter A.	Austin
Lommen, Peter A., Jr.	Austin
Longfellow, Helen W.	Brainerd
† Loomis, Earl A.	Minneapolis
Loomis, George L.	Winona
† Lorenc, Ernest	Rochester
Lorentzen, Ernest S.	Detroit Lakes
Lott, Frederick H.	Minneapolis
Louisell, Charles T.	Fairmont
Love, Frederick A.	Carlos
Love, J. Grafton	Rochester
Lovett, Beatrice R.	Oak Terrace
Lowe, Earl R.	So. St. Paul
Lowe, Thomas A.	So. St. Paul
Lowry, Elizabeth C.	Minneapolis
Lowry, Paul T.	Minneapolis
Lowry, Thomas	Minneapolis
† Luck, Hilda	Mankato
Luckemeyer, Carl J.	St. Cloud
Lueck, Wallace W.	Minneapolis
Lufkin, Nathaniel H.	Minneapolis
Lukk, Olaf	Montgomery
Lund, Carl J. T.	Fergus Falls
Lund, George W.	Minneapolis
Lund, Werner J.	Staples
Lundberg, Ruth I.	Minneapolis
Lundblad, Roy A.	Minneapolis
Lundblad, Stanley W.	Minneapolis
Lundeberg, Karl R.	Minneapolis
Lundell, Carl L.	Granite Falls
Lundholm, Arthur M.	Harris
Lundquist, Curt W.	Owatonna
Lundquist, Virgil J. P.	Minneapolis
Lundsten, Leslie C.	Bemidji
Lundy, John S.	Rochester
Lynch, Francis W.	St. Paul
† Lynch, Matthew J.	Minneapolis
Lyon, John D., Jr.	Hopkins
Lyons, James H.	Minneapolis
Lyons, Michael W.	Minneapolis
Lyons, William S.	Douglas
† Lysne, Henry	Minneapolis
Lysne, Myron	Robbinsdale
† Lysyj, Anatol	Minneapolis
Lyzenga, Anton G.	Minneapolis
† McAllister, James	Rochester
McBean, James B.	Rochester
McCabe, James S.	St. Paul
McCaffrey, F. John	Minneapolis
McCain, Donovan L.	St. Paul
McCammon, Joseph	Rochester
McCampbell, Malcolm D.	Minneapolis
McCannel, Malcolm A.	Minneapolis
McCarten, Francis M.	Stillwater
McCarthy, Austin M.	Willmar
McCarthy, Donald	St. Paul
McCarthy, Joseph J.	St. Paul
McCartney, James S.	Minneapolis
*†McCarty, Paul D.	Tower
† McClanahan, James H.	White Bear Lake
McClanahan, Thomas S.	White Bear Lake
McClary, George B., Jr.	Rochester
McClellan, Robert J.	St. Paul
McCloud, Charles N., Jr.	St. Paul
McClure, Rensselaer W., Jr.	Lawrence, Kansas
McConahey, William M., Jr.	Rochester
McCormick, Donald P.	Minneapolis
McCown, Louis K.	Memphis, Tenn.
† McCoy, Mary K.	Duluth
† McDaniel, Orianna	Minneapolis
McDaniel, S. P.	Lakeville
† McDonald, Archibald L.	Unknown
† McDonald, Colin C.	Rochester
McDonald, John R.	Rochester
McDonald, Owen G.	Duluth
† McDowell, John P.	St. Cloud
McEnaney, Clifford T.	Owatonna
McEwan, Alexander	St. Paul
McFarland, Arthur H.	Minneapolis
McGandy, Robert F.	Minneapolis
McGeary, George E.	Minneapolis
McGill, Leon K.	Virginia
McGoon, Dwight C.	Rochester
McGroarty, Brian J.	St. Paul
† McGroarty, John J.	Easton
McHaffie, Orval L.	Duluth
McHutchison, Samuel	St. Charles
McIlhany, Mary Lou	Lantana, Florida
McInerny, Maurice W.	Minneapolis
McIntyre, John A.	Owatonna
McKaig, Alan M.	Red Lake Falls
McKaig, Carle B.	Pine Island
McKay, Kenneth H.	Muscatine, Iowa
McKelvey, John L.	Minneapolis
McKenna, Jay K.	Austin
McKenna, John J.	Virginia
McKenna, Maurice J.	Grand Rapids
McKenzie, Charles H.	Minneapolis
McKenzie, Eva E.	St. Paul
McKinlay, Chauncey A.	Minneapolis
McKinlay, Gordon L.	Minneapolis
McKinney, Frank S.	Minneapolis
McLane, William O.	Brainerd
McLaughlin, Byron H.	Minneapolis
† McLaughlin, Edmund M.	Winona
† McMahan, Robert O.	Rochester
McManus, William F.	Princeton
McMurtrie, William B.	Minneapolis
McNear, George R., Jr.	Mankato
McNeil, John J.	Minneapolis
McNeil, Maurice R.	Glencoe
McNeill, J. A.	St. Paul
McNutt, John R.	Duluth
McPheeters, Herman O.	Minneapolis
† McQuarrie, Irvine	Minneapolis
MacCarty, Collin S.	Rochester
† MacCarty, William C.	Rochester
† MacDonald, Daniel A.	Minneapolis
MacDonald, John W.	Minneapolis
MacKinnon, Donald C.	Minneapolis
MacLean, Alexander R.	Rochester
MacLean, Lloyd D.	St. Paul
MacRae, Gordon C.	Duluth
Mach, Frank B.	Minneapolis
Mach, Ralph F.	Pine City
Macheledt, Neil L.	Anoka
Macklin, William E., Jr.	Willmar
Mackoff, Sam M.	Phoenix, Arizona
Madland, Robert S.	St. Paul
Maeder, Edward C.	Minneapolis
Magath, Thomas B.	Rochester
Magney, Fredolph H.	Duluth
Magnuson, Allen E.	Wheaton
Magnuson, Raymond C.	Cambridge
Magraw, Richard M.	Minneapolis
† Maher, Frank T.	Rochester
Mahle, Donald G.	Plainview
Mahle, James P.	Minneapolis
Maitland, Edwin T.	Jackson
Maks, Stephen W.	Rochester
† Maland, Clarence O.	Minneapolis
Malerich, J. Anthony	St. Paul
Malerich, J. Anthony, Jr.	St. Paul
† Malkasian, George D., Jr.	Rochester
Malmstrom, John A.	Virginia
Mandel, Sheldon L.	Minneapolis
Mankey, James C.	Minneapolis
Mankin, Harold T.	Rochester
Manlove, Charles H., Jr.	St. Paul
Mann, George A.	Minneapolis
† Manson, Frank M.	Worthington
‡ Margulis, Alexander R.	Fort Bragg, N. C.
Mark, Aaron L.	Minneapolis
Mark, Hilbert	Toledo, Ohio
Marking, George H.	Minneapolis
Marks, Roger W.	St. Paul
Marquis, John W.	Rochester
Marrone, Patrick H.	Duluth
Marshall, Clark M.	Crosby
Martens, Theodore G.	Rochester
Martin, Albert C.	Luverne

Martin, Dwight L. .... St. Paul
Martin, Frank E. .... Minneapolis
Martin, George B. .... Thief River Falls
Martin, George R. .... Minneapolis
Martin, Gordon M. .... Rochester
‡ Martin, Maurice J. .... San Juan, Porto Rico
† Martin, Thomas Philip .... Minneapolis
Martin, Webster C. .... Duluth
Martin, William B. .... Duluth
Martin, William J. .... Rochester
† Martineau, Joseph L. .... St. Paul
Martinson, Carl J. .... Wayzata
Martinson, Elmer J. .... Wayzata
Marvin, Joseph E. .... Brainerd
† Masson, Duncan M. .... Rochester
† Masson, James C. .... Rochester
Masson, James K. .... Rochester
Mast, Fredric L. .... Chisholm
† Mastman, Gary J. .... Rochester
Mateo, Guillermo .... St. Paul
Mathieson, Don R. .... Rochester
Matthews, James H. .... Minneapolis
* Mattill, Peter M. .... Oak Terrace
Mattison, Percy A. .... Winona
Mattson, Albert D. .... St. James
Mattson, Hamlin A. N. .... Minneapolis
Maunder, John B. .... Minneapolis
Maus, Philip W. .... Dawson
† Maxeiner, Stanley R. .... Minneapolis
Maxeiner, Stanley R., Jr. .... Minneapolis
† Mayberry, William E. .... Rochester
Mayne, John Gregory .... Rochester
Mayne, Roy M. .... Nopeming
Mayo, Charles W. .... Rochester
Mazzitello, William F. .... St. Paul
† Mead, Charles H. .... Duluth
* Meade, John R. .... St. Paul
Mears, Burtis J. .... St. Paul
* Mears, Robert F. .... Northfield
Medelman, John P. .... St. Paul
Meinert, Albert E. .... Winona
Meinert, John K. .... Willmar
Melancon, Joseph F. .... St. Paul
† Melby, Benedik .... Blooming Prairie
Meller, Maurice .... Brainerd
Meller, Robert L. .... Minneapolis
† Melzer, George R. .... Lyle
Mendelson, David F. .... Minneapolis
Menefee, Edward C. .... Albert Lea
Menold, William F. .... St. Paul
Mensheha, Nicholas .... Forest Lake
Mercil, William F. .... Crookston
Merkert, Charles E. .... Minneapolis
Merkert, George L. .... Minneapolis
Merner, Thomas B. .... Fairbault
† Merrick, Charlotte T. .... St. Paul
Merrick, Robert L. .... St. Paul
Merrill, Robert W. .... Morris
* Merriman, Lloyd L. .... Duluth
Merritt, Wallace A. .... Rochester
Messenheimer, Myron G. .... Minneapolis
Metcalf, Norman B. .... Princeton
Metz, Donald D. .... Buffalo Lake
Metzerott, Kirk O. .... Rochester
Meyer, Alvin J. .... Minneapolis
† Meyer, Anthony A. .... Minneapolis
† Meyer, E. Lawrence .... Minneapolis
Meyer, Frederick C. .... Kenyon
Meyer, Paul F. .... Faribault
Meyer, Robert J. .... Minneapolis
Meyer, Robert P. .... Faribault
† Meyerding, Edward A. .... St. Paul
† Meyerding, Henry Wm. .... Rochester
Michael, Joseph C. .... Minneapolis
Michel, Henry H. .... Minneapolis
Michels, Roger P. .... Willmar
Michelson, Henry E. .... Minneapolis
Michienzi, Leonard J. .... St. Paul
† Mickelsen, Emma F. .... Minneapolis
Mickelson, John C. .... Mankato
Midboe, Gilbert T. .... St. Paul
Middlebrook, John E. .... Minneapolis
Midthune, Andreen S. .... Lake Park
Miettunen, John B. .... Hibbing
Milhaupt, Emmett N. .... St. Cloud
Miller, Albert G. .... St. Paul
† Miller, Archie W. .... Rochester
Miller, Arden L. .... Minneapolis
Miller, Harold E. .... Minneapolis
Miller, Herman .... Austin
Miller, Hugo E. .... Minneapolis
Miller, John Carleton .... Minneapolis
Miller, Roland D. .... Rochester
Miller, Ross H. .... Rochester
† Miller, Victor I. .... Mankato
Miller, William P. .... Montevideo
Miller, William T. .... St. Paul
Miller, Winston R. .... Red Wing
Miller, Z. R. .... St. Paul
Millett, D. Keith .... Minneapolis
Millikan, Clark H. .... Rochester
Mills, John L. .... Winnebago
Mills, Stephen D. .... Rochester
Milnar, Frank J. .... St. Paul
Milroy, Thomas W. .... Des Moines, Iowa
Milton, John S. .... Minneapolis
Minge, Raymond K. .... Worthington
Minor, Ralph H. .... Rochester
Minsky, Armen A. .... Minneapolis
Mishek, Charles J. .... St. Paul
Mitby, Irvin L. .... Minneapolis
Mitchell, Berton D. .... Minneapolis
Mitchell, Edward C. .... Minneapolis
Mitchell, Mancel T. .... Minneapolis
Mixer, Harry W. .... Minneapolis
Moberg, Clarence W. .... Detroit Lakes
Mochal, Milo A. .... Thief River Falls
Moe, John H. .... Minneapolis
Moe, Thomas .... Moose Lake
Moehn, John T. .... Minneapolis
Moehring, Henry G. .... Duluth
Moen, Johannes K., Jr. .... Minneapolis
† Moersch, Frederick P. .... Rochester
Moersch, Herman J. .... Rochester
Moertel, Charles G. .... Rochester
Moga, John A. .... St. Paul
Molander, Herbert A. .... St. Paul
Molenaar, Robert E. .... Cannon Falls
Mollers, Theodore P. .... Soudan
Molnar, George D. .... Rochester
† Monahan, Elizabeth S. .... Minneapolis
Monahan, Robert H. .... St. Paul
Monserud, Nels O. .... Cloquet
Monson, Einer M. .... Minneapolis
Monson, Leonard J. .... Canby
Montgomery, Hamilton .... Rochester
Mooney, Robert D. .... St. Paul
Moore, Irvin H. .... Minneapolis
Moorhead, Marie .... Minneapolis
Moos, Daniel J. .... Minneapolis
† Moquin, Marie A. .... St. Paul
† Morehead, Dewey E. .... Owatonna
† Moren, J. Adelaide .... White Bear Lake
Morgan, Hugh O. .... Amboy
Moriarty, Berenice .... St. Paul
Moriarty, Cecile R. .... St. Paul
Morisaki, Michael M. .... Rochester
Mork, A. Harold .... Anoka
Mork, Frank E. .... Anoka
Morlock, Carl G. .... Rochester
Morrell, Frank .... Minneapolis
Morrison, Charlotte J. .... Minneapolis
Morrow, George W., Jr. .... Rochester
Mores, Morton P. .... LeRoy
Morsman, L. William .... Hibbing
† Mortensen, Nels G. .... Minneapolis
Mortenson, Howard O. .... Menahga
Mosby, Maurice E. .... Long Prairie
Moses, Royal R. .... Kenyon
† Moskowitz, Roland W. .... Rochester
Mosser, Donn G. .... Minneapolis
Moulton, Keith B. .... St. James
Mouritsen, Glenn J. .... Fergus Falls
Moyer, John B. .... Duluth
Moyer, Leonard B. .... Minneapolis
Mueller, Donald R. .... Bagley
Mueller, Rudolph B. .... Richmond
Muesing, William J. .... New Ulm
Muir, Walter F. .... Browns Valley
Mulder, Donald W. .... Rochester
Mulholland, William M. .... Minneapolis
Muller, A. Eugene .... No. St. Paul
Muller, John J. .... Hibbing
Muligan, Arthur M. .... Brainerd
Mundahl, Harold R. .... St. Paul
Munger, James E. .... Duluth
Munson, Martin S. .... Barnum
Murn, Thomas G. .... St. Cloud
Murphy, Edmund P. .... Minneapolis
Murphy, Jack T. .... St. Paul
Murphy, Joseph E. .... Marshall
Murray, Elisabeth M. .... Minneapolis
Murray, Robert A. .... Hibbing
Murtaugh, Robert J. .... Wadena
Musachio, Nicholas F. .... Foley
Muske, Marvin M. .... Minneapolis
Mussey, Mary E. .... Rochester
*†Mussey, Robert D. .... Rochester
Musty, Nicholas J. .... Minneapolis
Myers, Jay A. .... Minneapolis
Myers, John W. .... Canby
Myers, Thomas T. .... Rochester
Myhre, James .... Minneapolis
Myre, Clifford R. .... Paynesville

Nagel, Harold D. .... Minneapolis
Nagobads, Visvaldis J. .... Minneapolis
Nakamura, James Y. .... Deer River
Nash, Eldore B. .... Minneapolis
Nash, Leo A. .... St. Paul
Naslund, Ames W. .... Minneapolis
Navrital, Donald R. .... Glencoe
Neal, Joe M. .... St. Paul
Nealy, Donald E. .... Adrian
Neary, Richard P. .... Minneapolis
Neel, Harry B. .... Albert Lea
Neff, Walter S. .... Virginia
Nehring, Jesse P. .... Preston
Neibergs, Lidija .... St. Paul
Neibergs, Pauls .... St. Paul
† Neil, Robert L. .... Rochester
Neils, Vernon E. .... Sauk Rapids
Neiswanger, Walter E. .... Rochester
† Nelson, Bernette G. .... Hoyt Lakes
† Nelson, Bernice A. .... Minneapolis
Nelson, Carleton A. .... Minneapolis
Nelson, C. Barton .... Minneapolis
Nelson, Charles H. .... Albert Lea
Nelson, Clayton E. .... Albert Lea
Nelson, Edward N. .... Minneapolis
Nelson, Erland R. .... Minneapolis
Nelson, Ernest J. .... Lonsdale
Nelson, Glenn E. .... Fairfax
Nelson, Gunard A. .... Rochester
Nelson, Harvey .... Minneapolis
† Nelson, Henry E. .... Crookston
Nelson, Kenneth L. .... Texas
Nelson, Lloyd S. .... Minneapolis
Nelson, Loren E. .... St. Paul
† Nelson, Louis A. .... St. Paul
Nelson, Louis A., Jr. .... St. Paul
Nelson, Luther A. .... Rush City
Nelson, Maxine O. .... Minneapolis
Nelson, Maynard C. .... Minneapolis
Nelson, Melvin S. .... Granite Falls
*†Nelson, Nesmith P. .... Minneapolis
Nelson, O. L. Norman .... Minneapolis
Nelson, Robert H. .... Benson
Nelson, Roy A. .... Fergus Falls
Nelson, Wallace I. .... Minneapolis
Nelson, Wilburn O. B. .... Arizona
Nelson, Wilhma B. .... Albert Lea
Nerenberg, Sidney .... Minneapolis
Nesheim, Martin O. .... Emmons
Nessa, Curtis B. .... St. Cloud
Nesse, James A. .... Austin
Nesset, Lawren B. .... Minneapolis
Nesset, William D. .... Minneapolis
Neumaier, Arthur A. .... Glencoe
Neumann, Conrad A. .... Lewiston
Neumann, Roland F., Jr. .... Minneapolis
Neumeister, Charles A. .... Minneapolis
Newnum, Raymond L. .... Rochester
Niazi, Suad A. .... Clarkfield
Nice, Charles M. .... Minneapolis
Nichols, Donald R. .... Rochester
Nichols, Francis C. .... Woodridge, N. J.
Nichols, Raymond D. .... Crosby
† Nicholson, Murdoch A. .... Duluth
Nicholson, Richard W. .... Heron Lake
Nickerson, John R. .... Fairmont
Nickerson, Neil D. .... Fairmont
† Niehaus, Karl F. .... Rochester
Nielsen, Alvin M. .... Northfield
Nietfeld, Aloys B. .... Sauk Centre
Nimlos, Kenneth O. .... St. Paul
† Nimlos, Lenore O. .... St. Paul
Ninneman, Newton N. .... Waconia
Nisius, George F. .... Duluth
Nixon, James B. .... Crosby
Noble, John F. .... St. Paul
Noble, J. Lawrence .... St. Paul
† Nolan, Robert B. .... Rochester
Nolan, Robert K. .... Minneapolis
Nollet, Donald J. .... Hibbing
Noran, Axel S. N. .... Minneapolis
Noran, Harold H. .... Minneapolis
Norberg, Carl E. .... Cloquet
Nord, J. Erling .... Staples
Nord, Robert E. .... Minneapolis
† Nordin, Gustaf T. .... Saratoga, Calif.
Nordland, Martin .... Minneapolis
Nordland, Martin, Jr. .... Minneapolis
Nordlund, Mildred E. .... Cass Lake
Nordman, Willard F. .... Mora
Norman, David D. .... St. Paul
Norman, Mark L., Jr. .... Minneapolis
Normann, Stephen T., Jr. .... Waseca
Norquist, Joseph L. .... Thief River Falls
Norris, Neil T. .... Caledonia
† Norstrom, Craig W. .... Rochester
† Novak, Edward E. .... New Prague
Nuebel, Charles J. .... St. Paul
Nuessle, Walter G. .... Springfield
Nuessle, William F. .... Minneapolis
Nuetzman, Arthur W. .... Faribault
Nutting, Roland E. .... Duluth
Nydahl, Malvin J. .... Minneapolis
† Nydell, Carl C., Jr. .... Rochester
Nye, Katherine A. .... St. Paul
Nye, Lillian L. .... St. Paul
Nygren, William T. .... Braham
Nyhus, Ralph E. .... Hibbing
Nylander, Emil G. .... Minneapolis
Nywall, Dean D. .... Slayton

O'Brien, J. C.	St. Paul
O'Brien, Louis T.	Breckenridge
O'Brien, William A.	Minneapolis
† O'Connor, Daniel C.	Eden Valley
*†O'Connor, Loren J.	St. Paul
† O'Donnell, James E.	Minneapolis
O'Hanlon, John A.	Minneapolis
O'Kane, Thomas W.	St. Paul
O'Keefe, James P.	St. Cloud
O'Leary, John B.	Brainerd
O'Malley, Valentine	St. Paul
O'Neill, John C.	Duluth
O'Phelan, E. Harvey	Minneapolis
O'Reilley, Bernard E.	St. Paul
*†Oberg, Carl M.	Minneapolis
Ochsner, Clarence G.	Wabasha
Ockuly, Orville	St. Paul
Odel, Howard M.	Rochester
Odland, Donald M.	Luverne
Odland, Mark E.	Detroit Lakes
Odland, Olin M.	Granite Falls
Oeljen, Siegfried C. G.	Waseca
† Ogden, Warner	St. Paul
† Ohage, Justus	St. Paul
† Okihiro, Michael M.	Rochester
Olavs, Olga	Minneapolis
Olds, George H.	New Richland
Olfelt, Paul C.	Minneapolis
Olinger, John N.	St. Cloud
Olive, John T., Jr.	Mankato
Oliver, Irwin L.	Graceville
Oliver, James	Moorhead
Olmanson, Edmund G.	St. Peter
Olmanson, Myron D.	St. Peter
Olsen, Arthur M.	Rochester
Olsen, E. George	Minneapolis
Olsen, Jay R.	Minneapolis
Olsen, Ralph L.	St. Paul
Olson, Albert E.	Duluth
Olson, Albert J.	Owatonna
Olson, Alton C.	Minneapolis
Olson, Archie O.	Duluth
Olson, Carl J.	Minneapolis
Olson, C. Kent	Minneapolis
Olson, Charles A.	St. Paul
Olson, Chester J.	Belle Plaine
Olson, Detlof M.	Minneapolis
Olson, Duane O. C.	Gaylord
Olson, Ernest A.	Pine Island
Olson, Grant E.	West Concord
Olson, Gregory M.	Litchfield
Olson, Lillian A.	Ah-gwah-ching
† Olson, Olaf A.	Minneapolis
Olson, Philip A.	Minneapolis
Olson, Rolland A.	Wayzata
Olson, Robert T.	Canby
Onsgard, L. Kenneth	Houston
Oppegaard, Chester L.	Crookston
Oppen, E. Gerhard	Minneapolis
Oppen, Melvin G.	Wayzata
Opshal, Lawrence J.	Willmar
Opstad, Earl T.	Unknown
Orr, Burton A.	Faribault
Osborn, Donald O.	Austin
* Osborn, John E.	Rochester
Osmundson, Philip J.	Rochester
Ostergaard, Erling	Evansville
Ostergren, Edward W.	St. Paul
Ott, Eugene C.	Minneapolis
† Otto, Henry C.	Frazee
Ouellette, Alfred J.	St. Paul
Ourada, Anthony L.	Fairmont
Owen, Charles A., Jr.	Rochester
Owen, Richard R.	Minneapolis
Owens, Ben P.	Hibbing
Owens, Frederick M., Jr.	St. Paul
Owens, William A.	Montevideo
Ozolins, Marta	Faribault

Paal, Dwain J.	Minneapolis
Paciotti, Vincent J.	Hibbing
Page, Raymond L.	St. Charles
Palen, Benjamin J.	Minneapolis
Palm, Neil M.	St. Paul
Palmer, Clinton F.	Albert Lea
Palmer, Harry A.	Black Duck
† Pankratz, Peter J.	Taiwan (Formosa)
Papermaster, Ralph	Two Harbors
Papermaster, Theodore C.	Minneapolis
Paris, Jaime	Rochester
Park, Wilford E.	Minneapolis
Parker, Charles W.	Wadena
Parker, Harry L.	Rochester
Parker, Philip J.	Staples
Parker, Robert L.	Rochester
Parker, Warren E.	Sebeka
Parker, Wilbert H.	Chisholm
Parkhill, Edith M.	Rochester
Parkin, Thomas W.	Rochester
† Parr, Eugene Q.	Rochester
Parson, E. Irvine	Duluth
Parson, E. Lillian B.	Elbow Lake
Parson, Lester R.	Elbow Lake
Parsons, R. A.	St. James
Parsons, Ralph L.	Monterey
Pascuzzi, Chris A.	Rochester
Pasek, Antone W.	Cloquet
Pasek, Edward A.	Fairbault
Patch, Orien B.	Duluth
Patrick, Robert T.	Rochester
Pattee, James J.	Minneapolis
Patterson, Hugh D.	Slayton
† Patterson, William L.	Fergus Falls
Paulson, Elmer C.	Minneapolis
Paulson, John A.	Rochester
† Paulson, Theodore S.	Fergus Falls
Paulson, Wallace J.	Minneapolis
Payne, Richard E.	Virginia
Payne, William S.	Rochester
† Pearsall, R. P.	Virginia
Pearson, Bror F.	Shakopee
Pearson, Fritz R.	St. Paul
Pearson, Malcolm M.	St. Paul
Pease, Gertrude L.	Rochester
Peck, Willard R.	St. Paul
Pedersen, Arthur H.	St. Paul
Pedersen, Robert L.	Brainerd
Pedersen, Roy C.	Duluth
Peluso, Charles R.	Minneapolis
Pelzl, Charles R.	Pine River
† Pemberton, John D.	Rochester
Penk, Engward L.	Springfield
Penn, George E.	Mankato
† Pennie, Daniel F. V.	Duluth
† Peppard, Thomas A.	Minneapolis
Perkins, Douglass E.	Alexandria
Perlman, Everett C.	Minneapolis
Perlman, Herschel L.	Minneapolis
Perry, Harold	Rochester
Person, John P.	Albert Lea
Pertl, Albert L.	Canby
Peteler, Jennings C. L.	Minneapolis
Peterman, Albert F.	Rochester
Peters, Gustavus A.	Rochester
Petersen, Deane A.	Minneapolis
Petersen, Donald H.	Northfield
Petersen, Glenn L.	Minneapolis
Petersen, Magnus C.	Rochester
Petersen, Peter C.	Minneapolis
Petersen, Robert T.	St. Cloud
Petersen, William E.	Minneapolis
Peterson, Alvin C.	Mora
Peterson, David B.	St. Paul
Peterson, Donald B.	Anoka
Peterson, Donald H.	St. Paul
Peterson, Edward A.	St. Paul
Peterson, Edward N.	Virginia
Peterson, Harold O.	St. Paul
Peterson, Henry	Minneapolis
Peterson, Herbert W.	Minneapolis
Peterson, Joel L. E.	St. Paul
Peterson, John H.	Duluth
Peterson, Kenneth A.	Marshall
Peterson, Kenneth H.	Hutchinson
Peterson, Lowell F. A.	Rochester
Peterson, Nordahl P.	Minneapolis
Peterson, Oliver H.	Minneapolis
Peterson, Oliver H., Jr.	Minneapolis
Peterson, Palmer A.	Minneapolis
Peterson, Paul A., Jr.	Rochester
† Peterson, Peter E.	Unknown
Peterson, Roy A.	Vesta
Peterson, Roy L.	White Bear Lake
Peterson, Stanley C.	Austin
Peterson, W. H.	Austin
Peterson, Willard C.	Minneapolis
Peterson, Willard E.	Willmar
Petit, Julien V.	Minneapolis
† Petit, Leon J.	Minneapolis
Pettersen, George R.	Aitkin
‡ Pettet, John R.	Laguna Beach, Calif.
Pewters, John T.	Minneapolis
Peyton, William T.	Minneapolis
Phalen, Patrick T.	Rochester
Phares, Otto C.	St. Cloud
† Phelps, Kenneth A.	Menlo Park, Calif.
Philp, David R.	Watertown
Pierce, Charles H.	Wadena
Pierce, Jack R.	Wiiliamson, W. Va.
Pierce, Robert B.	Renville
Pierson, Roy F.	Slayton
† Piper, Monte C.	LaCanada, Calif.
† Piper, William A.	Mountain Lake
Plasha, Matthew K.	Anoka
Plass, Herbert F. R.	Minneapolis
† Platou, Erling S.	Minneapolis
Pleissner, Karl W.	Minneapolis
Plimpton, Nathan C., Jr.	Minneapolis
Plotke, Harry L.	St. Paul
Plott, Carol L.	Heron Lake
Plucker, Milton W.	Worthington
* Pogue, Richard E.	Carson City, Nevada
Pohl, John F. M.	Minneapolis
† Poirier, Joseph A.	Forest Lake
Pollak, Kurt	Minneapolis
Pollard, William S.	Duluth
Polley, Howard F.	Rochester
Pollock, Anthony J.	Minneapolis
† Pollock, David K.	Minneapolis
Polski, Paul G.	So. St. Paul
Polzak, Jacob A.	Minneapolis
Pone, John	Cambridge
Ponterio, James E.	Shakopee
Pool, Thomas L.	Rochester
Popadiuk, Peter	Minneapolis
† Poppe, Frederick H.	Coral Gables, Fla.
† Porter, Oliver M.	Atwater
Posey, John W.	Rochester
Post, Edmund A.	St. Paul
Potter, Robert B.	Minneapolis
Potts, L. C.	Warroad
Power, John E.	Duluth
Power, John E., Jr.	Duluth
† Pratt, Fred J.	Minneapolis
Pratt, Fred J., Jr.	Minneapolis
Pratt, Joseph H., Jr.	Rochester
† Preine, Irving A.	Osseo
† Preisinger, Joseph W.	Dallas, Texas
Prem, Konald A.	Minneapolis
Preston, Frank S., Jr.	Minneapolis
Preston, Paul J.	Minneapolis
Price, William E.	Minneapolis
Prickman, Louis E.	Rochester
Priest, Robert E.	Minneapolis
Priestley, James T.	Rochester
† Prim, Joseph A.	Minneapolis
Prins, Leo R.	Albert Lea
Prlina, Isaac M.	Virginia
Proeschel, Ray K.	Willmar
Proffitt, William E.	Minneapolis
* Proshek, Charles E.	Minneapolis
Proud, Harry S.	St. Paul
Pruitt, Raymond D.	Rochester
Pugh, David G.	Rochester
Pumala, Erven E.	Warren
Purnell, Don C.	Rochester
Purves, G. Harland	Buffalo
Puumala, Reino H.	Cloquet

Quanstrom, Virgil E.	Brainerd
Quattlebaum, Frank W.	St. Paul
Quello, Robert O. B.	Minneapolis
† Quetsch, Richard M.	Rochester
Quiggle, Arthur B.	Minneapolis
Quist, Henry W., Jr.	Minneapolis

Raaen, Olaf	Minneapolis
Raattama, John W.	Nashwauk
Racer, Harley J.	Minneapolis
Raetz, Sylvester J.	Maple Lake
Ragen, Patrick A.	Rochester
Raile, Richard B.	Minneapolis
Rajala, Arnold I.	Grand Rapids
Ralph, James R.	St. Paul
Ralston, Donald E.	Rochester
Ramlow, Ralph M.	St. Paul
Ramsdell, John A.	Rochester
† Ramsey, Walter R.	St. Paul
Randall, Lawrence M.	Rochester
Randall, Raymond V.	Rochester
Ransom, H. Robert	Osseo
† Ransom, Matthias L.	Hancock
Rasmussen, Ramby C.	St. Paul
Ratelle, Alexander E.	Minneapolis
Ravits, Harold G.	St. Paul
Rayner, Ralph R.	St. Paul
Rea, Charles E.	St. Paul
* Reader, Donald R.	Minneapolis
Reardon, Andrew E.	Duluth
‡ Redding, Foster K.	Minneapolis
Redford, John B.	Rochester
Reed, Henry H.	Duluth
Reed, Paul	Virginia
Reed, Sheldon C.	Minneapolis
Reef, Alan R.	Crookston
Regan, John J.	Minneapolis
Regnier, Edward A.	Minneapolis
Reichelderfer, Charles F.	Staples
Reichelt, Leland G.	Duluth
Reid, James W.	So. St. Paul
Reif, Harold A.	Minneapolis
Reif, Henry J.	St. Cloud
Reif, Robert W.	White Bear Lake
‡ Reifsnyder, William Henry, III	Rochester
Reiley, Richard E.	Minneapolis

*†Reineke, George F.	New Ulm
Reinhardt, James H.	Alexandria
Reiser, Milton P.	St. Louis Park
Reitemeier, Richard J.	Rochester
ReMine, William H., Jr.	Rochester
Remole, William D.	Minneapolis
Remsberg, Robert R.	Tracy
† Replogle, William H.	Los Angeles, Calif.
Resch, Joseph A.	Minneapolis
Restall, Charles J.	Rochester
Reynolds, Donald P.	Minneapolis
† Reynolds, William A.	Rochester
Rholl, Arnold O.	Minneapolis
Rice, Carl O.	Minneapolis
Rice, Frank B.	Minneapolis
Rice, Fred A.	Minneapolis
Rice, Hagbart G.	Moorhead
Richards, Albert M.	St. Paul
Richards, Ernest T. F.	St. Paul
Richards, William B.	St. Cloud
Richardson, Edward J., Jr.	St. Paul
Richardson, Robert J.	St. Paul
Richdorf, Lawrence F.	Minneapolis
Richter, David J.	Virginia
Rick, Paul F. W.	St. Paul
Rieke, Wellington W.	Wayzata
Rieschl, Elizabeth K.	Jordan
Rimas, Matthew J.	Comfrey
Ringle, Otto F.	Walker
Rinkey, Eugene	St. Paul
Riordan, Elsie M.	Minneapolis
Ripple, Rudolph J.	St. Paul
Risch, Ronald E.	Minneapolis
Risser, Alden F.	Stewartville
Ritchie, Wallace P.	St. Paul
Ritt, Albert E.	St. Paul
‡ Ritzinger, Frederick R.	Gowrie, Iowa
Rizer, Dean K.	Minneapolis
† Rizer, Robert I.	Wayzata
Roach, Donald E.	St. Paul
Roan, Morton	Lamberton
Robb, Edwin F.	Minneapolis
† Robbins, Charles P.	Winona
Robbins, Mason C.	St. Paul
Robbins, Owen F.	Minneapolis
Roberts, Byron H.	Minneapolis
† Roberts, Frank E.	Rochester
Roberts, Lewis J.	Minneapolis
Roberts, Oliver W.	Owatonna
Roberts, Stanley W.	Minneapolis
*†Roberts, William B.	Minneapolis
Robertson, Paul A.	Austin
Robilliard, Charles M.	Fairbault
Robinett, Robert W.	Worthington
Robinson, Cortland O.	Minneapolis
Robinson, David B.	Rochester
Robinson, Hugh P.	Rochester
Rock, William H.	Anoka
Rocknem, Robert E.	Minneapolis
Rockwell, Curtiss V.	Minneapolis
Rockwood, Philo H.	Fergus Falls
† Rodda, Frederick C.	Minneapolis
Rodgers, Richard S.	Minneapolis
Roehlke, Arthur B.	Elk River
Roemer, Henry J.	Winona
Rogers, Charles W.	Winona
* Rogers, Sydney F.	St. Paul
Rogin, Norton	Minneapolis
Rogne, William G.	Spring Grove
Roholt, Christian L.	McIntosh
Roholt, H. B.	Fosston
Rohrer, Christian A.	Worthington
Rolig, David H.	St. Paul
Rollie, Orris O.	St. Paul
Rollins, Pat	Wayzata
† Rollins, Troy G.	Rochester
Rome, Howard P.	Rochester
Romness, Kenneth B.	Mound
Rondeau, Eugene T.	Two Harbors
Rooke, Edward D.	Rochester
Rorem, Joseph A.	Appleton
Rose, John T.	Lakefield
Rosenbaum, David L.	Minneapolis
Rosendahl, Frederick G.	Minneapolis
Rosenfield, Abraham B.	Minneapolis
Rosenow, John H.	Minneapolis
Rosenthal, F. Harold	Austin
Rosenthal, Robert	St. Paul
† Rosenwald, Reuben M.	Coon Rapids
Ross, Alexander J.	Minneapolis
Ross, Griff T.	Rochester
† Ross, James V., Jr.	Rochester
† Ross, Margaret	Rochester
Rossberg, Raymond A.	Morris
Rossen, Ralph X.	Minneapolis
Rossing, Robert G.	St. Paul
Rotenberg, Robert J.	Minneapolis
Roth, Frederick D.	Mankato
Roth, George C.	St. Paul
‡ Roth, Harry L.	Carlstad, Calif.
Rothnem, Morris S.	Minneapolis
† Rothschild, Harold J.	St. Paul
Roust, Henry A.	Montevideo
Rovelstad, Randolph A.	Rochester
† Rovelstad, Roger A.	Yonkers, N. Y.
Rowe, Clarence J., Jr.	St. Paul
† Rowe, Olin W.	Duluth
Rowles, Everett K.	Coleraine
Roy, Phil C.	St. Paul
Ruchie, Warren H.	Willmar
Rucker, Charles W.	Rochester
Rucker, William H.	Minneapolis
Rud, Norman E.	Minneapolis
† Rudell, Gustave L.	Minneapolis
Rudie, Clifford N.	St. Peter
Rudie, Peter S.	Duluth
Rudie, William D.	Duluth
Ruggles, George M.	Forest Lake
† Ruhberg, George M.	Santa Barbara, Calif.
Rukavina, John G.	St. Paul
Rumpf, Carl W.	Faribault
Runquist, John M.	Duluth
Runquist, Richard K.	Good Thunder
Rushing, Lige B., Jr.	Rochester
Rushton, Joseph G.	Rochester
Russ, Homer H.	Blue Earth
† Russell, Marion F., Jr.	Rochester
Russeth, Arthur N.	Minneapolis
Rusten, Elmer M.	Minneapolis
Rusterholz, Alan P.	St. Paul
Rutledge, John B.	Detroit Lakes
Rutledge, Lloyd H.	Detroit Lakes
Ryan, John J.	St. Paul
Ryan, Joseph M.	St. Paul
Ryan, William J.	Duluth
Rydburg, Wayne C.	Minneapolis
Ryding, Vincent	Mountain Lake
Rydland, Arne D.	Minneapolis
Rygh, Harold N.	Atwater
Rynda, Edwin R.	New Prague
Rynearson, Edward H.	Rochester
Rysgaard, George N.	Northfield
St. Cyr, Harry M., Jr.	Robbinsdale
St. Cyr, Kenneth J.	Robbinsdale
Sach-Rowitz, Alvan	Moose Lake
Sadler, William P.	Minneapolis
Saffert, Cornelius A.	New Ulm
‡ Safirescu, Sorin R.	San Francisco, Calif.
Sager, John H.	Rochester
† Sager, Stanley M.	Rochester
Sahr, Walter G.	Hutchinson
Salassa, Robert M.	Rochester
Saliterman, Bernard I.	Minneapolis
Salk, Richard J.	Albany
Salter, Reginald A.	Virginia
Samuelson L. Gordon	St. Peter
* Samuelson, Samuel	Minneapolis
Sanchez, James J.	Rochester
Sandeen, Robert M.	Buffalo
† Sanderson, Anton G.	Ashby
Sanderson, David J.	Fergus Falls
Sandt, Karl E.	Minneapolis
† Sandven, Nels O.	Paynesville
† Sanford, Arthur H.	Rochester
Sanford, John B.	Duluth
Sanford, Raymond A.	Mankato
† Sarff, Oliver E.	Duluth
Sargent, Edward C., Jr.	Austin
Sarnecki, M. M.	St. Paul
† Satersmoen, Theodore	Pelican Rapids
Sather, Edgar L.	Fosston
Sather, Edgar R.	Alexandria
Sather, George A.	Fosston
Sather, Richard N.	Fosston
Sather, Russell O.	Crookston
Satterlee, Howard W.	Lewiston
† Satterlund, Victor L.	New Richmond, Wis.
Sauer, William G.	Rochester
† Savage, Francis J.	St. Paul
Sawaryniuk, Iwan	Waconia
Sawatzky, William A.	Minneapolis
Sawtell, Robert R.	Worthington
Sax, Milton H.	Duluth
Sax, Simon G.	Duluth
Saxman, Gertrude O.	Georgetown
Sayre, George P.	Rochester
Sborov, Abe M.	Minneapolis
Scallen, Raymond W.	Minneapolis
Scanlon, Paul W.	Rochester
Schaaf, Frederick H. K.	Minneapolis
Schaar, Francis E.	Minneapolis
Schade, Frederick L.	Worthington
Schadt, Daniel C.	Rochester
Schaefer, Joseph C.	Rochester
Schaefer, Joseph F.	Owatonna
Schaefer, Kenneth F.	Minneapolis
Schaefer, Wesley G.	Minneapolis
Schaffhausen, Irwin F.	Minneapolis
Schaffhausen, Mildred	Minneapolis
Schamber, Walter F.	Parkers Prairie
*†Schatz, Francis J.	St. Cloud
Scheidel, Alois M.	Mankato
Scheifley, Charles H.	Rochester
† Scheldrup, N. H.	Minneapolis
Scherer, L. Raymond	Minneapolis
Scherling, Sidney S.	Minneapolis
Scheuneman, Allen F.	Warroad
Schiele, Burtrum C.	Minneapolis
Schimelpfenig, George T.	Chaska
Schirber, Martin J.	Grand Rapids
Schirger, Alexander	Rochester
Schissel, Gregory A.	Minneapolis
Schmid, John F.	Duluth
Schmidt, Herbert W.	Rochester
Schmidt, Hilmar R.	Rushford
† Schmidt, Paul A.	Aurora, Ill.
Schmidt, Paul G.	Granite Falls
Schmidt, Ruben F.	Alden
Schmidt, W. Robert	Minneapolis
Schmidtke, Reinhardt L.	St. Paul
† Schmitt, S. C.	Los Angeles, Calif.
Schmitz, Anthony A.	Mankato
Schmitz, Everett J.	St. Cloud
Schnabel, Robert F.	Crookston
Schneck, Jack I.	Minneapolis
Schneider, James A.	Rochester
Schneider, Lawrence E.	Duluth
Schneider, Paul J.	Adams
Schnell, Frederick S.	Litchfield
† Schoch, Robert B. J.	St. Paul
Schoeneberger, P. B.	Perham
Scholl, Virginia M.	Rochester
† Scholpp, Otto W.	Hutchinson
Scholz, Donald A.	Rochester
Schons, Edward	St. Paul
Schossow, George W.	Erskine
Schottler, Max E.	Minneapolis
† Schroder, Charles H.	Pottstown, Pa.
Schroeckenstein, Hugo F.	St. Paul
Schroeder, Albert J.	Minneapolis
Schroeppel, John E.	Winthrop
Schulberg, Verne A.	Gaylord
† Schuldt, Frederick C.	St. Paul
Schultz, Alvin L.	Minneapolis
Schultz, Earl A.	Minneapolis
† Schultz, J. Albert	Albert Lea
Schultz, J. Harold	Minneapolis
Schultz, Peter J.	Minneapolis
Schultz, Robert W.	Fairmont
† Schulze, Albert G.	St. Paul
Schulze, William M.	Minneapolis
Schumacher, John W.	Minneapolis
Schut, John W.	Anoka
Schutz, Elmer S.	Mountain Lake
Schwartz, Carl A.	Anoka
Schwartz, E. Robert	Minneapolis
† Schwartz, John T.	Rochester
Schwartz, Virgil J.	Minneapolis
Schweiger, Theodore R.	Hibbing
Schwyzer, Hanns C.	St. Paul
Schwyzer, Marguerite	St. Paul
Scott, Eugene E.	St. Paul
Scott, Horace G.	Minneapolis
Scott-Miller, James R.	Rochester
Scudamore, Harold H.	Rochester
† Seaberg, John A.	Pequot Lakes
Seashore, Rosel T.	Duluth
Seay, James Elbert, III	Rochester
‡ Sebrechts, Paul	Great Lakes, Ill.
Seery, Thomas M.	Austin
Segal, Martin A.	Minneapolis
Seham, Max	Minneapolis
Seifert, Milton H.	Excelsior
Seifert, Otto J.	New Ulm
Sekanina, Jan	Babbitt
Sekhon, Mohan S.	St. Paul
Selck, Wolfgang T.	Fergus Falls
Seldon, Thomas H.	Rochester
Sells, Richard J.	No. St. Paul
Selmo, Joseph D.	Norwood
† Semler, Herbert J.	Rochester
Semsch, Robert D.	Minneapolis
† Senn, Edward W.	Owatonna
Sethre, Arthur E.	Fergus Falls
Setzer, Hobert J.	St. Paul
Shandorf, James F.	Minneapolis
Shannon, William R.	St. Paul
Shaperman, Eva P.	Minneapolis
Shapiro, Irving	Minneapolis
Shapiro, Sidney K.	Minneapolis
Sharp, David V.	Minneapolis
Shaver, Ward	Fergus Falls
Shaw, Howard A.	Minneapolis
Shea, Andrew W.	Minneapolis
Sheedy, Chester L.	Austin
Shelander, Marcus I.	St. Paul
† Shellman, John L.	Pacific Palisades, Calif.
Sheppard, Charles G.	Hutchinson
Sher, David A.	Virginia
Sher, Lewis	Minneapolis
Sherman, Alfred G.	Albert Lea
Sherman, Carnot H.	Bayport
Sherman, Charles L.	Luverne

Sherman, Lloyd F.	Minneapolis
Sherman, Morris H.	Minneapolis
Sherman, Royal V.	Red Wing
† Sherrick, Donald W.	Rochester
*†Sherwood, George E.	Kimball
Schick, Richard M.	Rochester
Shillington, Maurice A.	Brainerd
Short, Jacob	St. Paul
Shragg, Robert I.	Minneapolis
Shronts, John F.	Minneapolis
Sidell, Franklin D.	Minneapolis
Siegel, Clarence	St. Paul
Siegel, John S.	Virginia
Siegmann, William C.	Minneapolis
Siekert, Robert G.	Rochester
Silas, Ralph M.	Minneapolis
Silver, Arthur W.	Rochester
Silver, John D.	Minneapolis
‡ Silvis, Stephen E.	San Francisco, Calif.
Simison, Carl	Barnesville
Simmonds, Harry N.	Prior Lake
Simons, Bernard H.	Chaska
Simons, Edwin J.	Edina
† Simons, Jalmar H.	Unknown
Simons, Leander T.	St. Paul
Simonson, Donald B.	Minneapolis
Simonton, Kinsey M.	Rochester
† Simpson, Ellery De W.	Asheville, N. Car.
Sinamark, Andrew	Hibbing
Singer, Benjamin J.	St. Paul
Sinykin, Melvin B.	Minneapolis
Siperstein, David M.	Minneapolis
Sisk, Harvey E.	St. Cloud
Sisler, Clifford E.	Minneapolis
*†Sivertsen, Andrew	Minneapolis
Sjoding, J. Donald	Mankato
Sjostrom, Lawrence E.	St. Peter
Skaife, William F.	Little Falls
Skaug, Harold M.	Chatfield
Skinner, Abbott	St. Paul
† Skinner, Harvey O.	St. Paul
Skjold, Arthur C.	Minneapolis
Skogerboe, Rudolph B.	Karlstad
† Skworcow, George	St. Paul
Slater, Sidney A.	Worthington
Smiley, John T.	Minneapolis
Smisek, Elmer A.	St. Paul
Smisek, Frank M.	Minneapolis
Smith, Adam M.	Minneapolis
Smith, Archie M.	Minneapolis
Smith, Baxter A., Jr.	Minneapolis
Smith, Cyril M.	Duluth
Smith, Don V.	Blue Earth
† Smith, Frederick L.	Rochester
Smith, George R.	Hutchinson
Smith, Graham G.	Minneapolis
Smith, Harry J.	Lake Crystal
† Smith, Harry L.	Rochester
*†Smith, Homer R.	Minneapolis
‡ Smith, John H.	Augusta, Ga.
† Smith, Lester L., Jr.	Rochester
Smith, Lucian A.	Rochester
† Smith, Margaret I.	Gardena, Calif.
† Smith, Myron W.	Red Wing
Smith, Nadine G.	Minneapolis
Smith, Norvin R.	Willmar
Smith, Paul M.	Lake Crystal
Smith, Ralph E.	Minneapolis
Smith, Reginald A.	Rochester
Smith, Theodore S.	Minneapolis
Smith, Thorsten	Faribault
Smith, Vernon D. E.	St. Paul
Smith, Wallace R.	Grand Marais
Smith, William G.	Rochester
Smith, William T.	Minneapolis
Smollen, William J.	Rochester
Smorstok, Matthew B.	Monticello
Smyth, John J.	Lester Prairie
Snider, Howard R.	Mankato
Snyder, Clifford D.	Kiester
Snyder, George W.	St. Paul
Snyker, Omer E.	Ely
Soderberg, Richard J.	Grand Marais
Soderlind, Regnar T.	Minneapolis
*†Sogge, Ludwig L.	Windom
Sohlberg, Olof I.	St. Paul
Solhaug, Samuel B.	Minneapolis
Solhaug, Samuel B., Jr.	Minneapolis
† Solsem, Frederick N. S.	Spicer
Solvason, Harold M.	Minneapolis
Sommerdorf, Vernon L.	St. Paul
Sommers, Ben	St. Paul
Sommerville, Robert L.	Rochester
Sonnesyn, Nels N.	Le Sueur
Sontag, David W.	Lake City
Sorem, Milton B.	St. Paul
Soucheray, Philip H.	St. Paul
Soule, Edward H.	Rochester
Souster, Benjamin B.	St. Paul
Sowada, Ernest J.	St. Paul
Spain, W. Thomas	Richmond, Va.
Spang, Anthony J.	Duluth
Spang, James S.	Duluth
Spang, William M.	Duluth
Spano, Joseph P	Minneapolis
Spencer, Bernard J.	Minneapolis
Spencer, Robert J.	Rochester
Sperl, Michael P., Jr.	Rochester
Spieler, Forrest B.	Pequot Lakes
Spink, Wesley W.	Minneapolis
Spittel, John A., Jr.	Rochester
Sponsel, Kenath H.	Minneapolis
Sprafka, Gregory A.	St. Paul
Sprafka, Joseph L.	St. Paul
* Sprafka, Joseph M.	St. Paul
Sprague, Randall G.	Rochester
† Spratt, Charles N.	Minneapolis
Spudis, Edward V.	Rochester
† Spurgeon, Frederick C.	Rochester
Spurzem, Raymond J.	Anoka
Stadem, Clifford J.	Twin Valley
Stahl, George W.	Austin
Stahler, Paul A.	Jordan
Stahr, Aubrey C.	Hopkins
Stam, John	Worthington
Stanford, Charles E.	Minneapolis
† Stangl, Philip E.	St. Cloud
† Stanley, Court R.	Minneapolis
Starekow, Milton D.	Thief River Falls
Starr, Grier F.	Rochester
† Starr, Jason L.	Rochester
Staub, Henry P.	Minneapolis
Stauffer, Maurice H.	Rochester
Steffens, Leon A.	Red Wing
Stein, Raymond J.	Pierz
Stein, William A.	Duluth
Steinberg, Charles L.	St. Paul
Steiner, Leon E.	Albert Lea
Steinhilber, Richard M.	Rochester
Stelter, Lloyd A.	Minneapolis
Stemsrud, Harold L.	Alexandria
Stennes, John L.	Minneapolis
Stensgaard, Kermit L.	Thief River Falls
† Stenstrom, Annette	Marine-on-the-St. Croix
Stephens, William E.	Minneapolis
Sterner, Donald C.	St. Paul
Sterner, E. R.	St. Paul
Sterner, John J.	St. Paul
Sterrie, Norman A.	Minneapolis
Stevenson, Basil M.	Fulda
Stewart, Alexander	St. Paul
Stewart, Donald E.	Crookston
Stewart, Marvin J.	Minneapolis
Stewart, Rolla I.	Minneapolis
Stickney, J. Minott	Rochester
Stiegler, Farrell S.	Minneapolis
Stillwell, George K.	Rochester
Stillwell, Walter C.	Mankato
Stilwell, George G.	Rochester
Stoesser, Albert V.	Minneapolis
Stolpestad, Armer H.	St. Paul
* Stolpestad, Herbert L.	St. Paul
Stoltz, Robert C.	Minneapolis
† Stoltze, Cynthia A.	Rochester
† Stomel, Joseph	Los Angeles, Calif.
Stone, Norman F.	Minneapolis
Stone, Stanley P.	Minneapolis
† Stoll, Newsom	Rochester
Storino, Henry E.	Rochester
Stormont, James R.	Rochester
Storsteen, Kenneth A.	Duluth
Stoy, Robert A.	Little Falls
*†Strauchauer, Arthur C.	Minneapolis
Strand, Jack W.	St. Paul
Stransky, Theodore W.	Owatonna
Strate, Gordon E.	St. Paul
Strathern, Carleton S.	St. Peter
† Strathern, Fred P.	St. Peter
† Strathern, Moses L.	Gilbert
Stratte, Alf K.	Pine City
Stratte, Harold C.	Windom
Strauchler, Jonas	Belview
Straus, M. L.	St. Paul
Street, Bernard	Northfield
Streitz, John M.	Duluth
Strem, Edward L.	St. Paul
Strewler, Gordon J.	Duluth
Strickler, Jacob H.	Minneapolis
* Strobel, William G.	Duluth
Stroebel, Charles F., Jr.	Rochester
Strom, Gordon W.	Minneapolis
Stromgren, Delph T.	Minneapolis
Stromme, William B.	Minneapolis
Strunk, Clarence A.	Minneapolis
Studer, Donald J.	Faribault
Stuhr, John W.	Stillwater
Sturges, Robert L.	Minneapolis
Sturley, Rodney F.	St. Paul
Subak, Barbara H.	Minneapolis
† Subby, Walter	Wayzata
Sukov, Marvin	Minneapolis
Sullivan, Charles R.	Rochester
Sullivan, Raymond M.	Minneapolis
Sullivan, William A., Jr.	Minneapolis
Summar, M. Thomas	Virginia
Sutherland, Harry N.	Ely
Sutherland, W. H.	Spicer
* Sutton, Harris R.	Hoffman
Svien, Hendrik J.	Rochester
Swain, Francis M.	Illinois
† Swanson, John A.	St. Paul
Swanson, Lawrence J.	St. Paul
Swanson, Roy E.	Minneapolis
Swanson, Wallace L.	Minneapolis
Swedberg, William A.	Duluth
Swedenburg, Paul A.	Glenwood
Sweetser, Horatio B.	Minneapolis
Sweetser, Theodore H.	Minneapolis
Sweetser, Theodore H., Jr.	Minneapolis
† Sweitzer, Samuel E.	Minneapolis
† Swendseen, Carl G.	Minneapolis
Swendseen, Carl J.	Graceville
Swendson, James J.	St. Paul
Swenson, Arnold O.	Duluth
Swenson, Donald B.	Mankato
Swenson, Donald B.	St. Paul
Swenson, Orvie J.	Waseca
Swenson, Richard W.	Hibbing
Swenson, Roy G.	North Branch
Symmonds, Richard E.	Rochester
Syverton, Jerome T.	Minneapolis

Tainter, Ronald W.	Marshall
Tam, Ernest C.	Minneapolis
† Tama, Lawrence	Rochester
Tangen, George M.	Minneapolis
Tani, George T.	St. Paul
Tanquist, Edwin J.	Alexandria
† Taswell, Howard F.	Rochester
Taylor, Joseph H.	Minneapolis
Taylor, William E.	Minneapolis
Teeter, Richard R.	Minneapolis
Teich, Kenneth W.	Duluth
Teisberg, John E.	St. Paul
Tenner, Robert J.	Minneapolis
Terrell, Bernard J.	Nopeming
Tesar, Charles E.	Unknown
Tesch, Gordon H.	Elk River
Testor, James V.	Winona
Tetlie, James P.	Duluth
Textor, Jerome	Anoka
Thal, Alan P.	Minneapolis
Thayer, Ellsworth A.	Fairmont
Theye, Richard A.	Rochester
Thielen, Robert D.	St. Michael
Thiem, Chester E.	Mankato
Thill, Leonard J.	Balaton
*†Thomas, George E.	Minneapolis
Thomas, John V.	Duluth
Thomas, William H.	Howard Lake
Thomes, A. Boyd	Minneapolis
Thompson, A. Henry	St. Peter
Thompson, Arthur	Minneapolis
Thompson, Carl O.	Hendricks
Thompson, Floyd A.	St. Paul
Thompson, Gershom J.	Rochester
† Thompson, John W., III	Rochester
Thompson, Russell A.	Cosmos
Thompson, Willis H.	Minneapolis
Thomson, James M.	Austin
Thoreson, M. C. Bernice	So. St. Paul
Thorsen, David S.	Minneapolis
Thorson, Stuart V.	Minneapolis
Thouin, Laurence G.	Hibbing
Thuringer, Carl B.	St. Cloud
Thysell, Desmond M.	Minneapolis
† Thysell, Fred A.	Moorhead
Thysell, Harold R.	Crookston
Thysell, Vernon D.	Hawley
Tiffany, Francis B.	St. Paul
Tifft, Cyril R.	St. Paul
Tillisch, Jan H.	Rochester
Tinkham, Robert G.	Minneapolis
Titrud, Leonard A.	Minneapolis
Tobin, John D.	Minneapolis
Tomhave, Wesley G.	Hibbing
Tongen, Lyle A.	St. Paul
Toon, Robert W.	Minneapolis
Torghele, John R.	Hastings
Tosseland, Noel E.	Duluth
Trach, Benedict B.	Minneapolis
Traeger, Carl A.	Faribault
Trautmann, James C.	Duluth
Travis, James S.	St. Paul
Traxler, J. Felix	Henderson
Tregilgas, Harold R.	So. St. Paul
Tregilgas, Richard B.	St. Paul
Troost, Henry B.	Mankato
Trow, James E.	Minneapolis
Trow, William H.	Minneapolis
Trueman, Harold S.	Minneapolis
Truesdale, Clark W.	Glencoe
Tsai, Shih Hao	Oak Terrace
Tucker, Richard C.	Minneapolis
Tudor, Richard B.	Minneapolis
‡ Tuffanelli, Denny L.	New York, N. Y.
† Tuohy, Edward L.	Santa Barbara, Calif.
† Turnacliff, Dale D.	St. Paul
Turner, John C., Jr.	Rochester

Tuura, James L.	Duluth
Tweedy, John A.	Winona
Tweedy, Robert B.	Winona
Twidwell, Joseph E.	Minneapolis
Twiggs, Leo F.	Austin
Twomey, John E.	Minneapolis
Tyler, Donald E.	Rochester

Ubel, Frank A.	St. Paul
Uhley, Charles G.	Crookston
Uihlein, Alfred	Rochester
Ulrich, Emery E.	Crosby
† Ulrich, Henry L.	Minneapolis
Ulvestad, Harold S.	Minneapolis
Underdahl, Laurentius O.	Rochester
† Undine, Clyde A.	Minneapolis
* Urberg, Sofus E.	Duluth
Utendorfer, Robert W.	Minneapolis
Utz, David C.	Rochester
Utz, Philip H.	La Crescent

Vaaler, Robert T.	Cokato
† Vadheim, Alfred L.	Tyler
† Valentine, Walter H.	Tracy
Van Bergen, Frederick H.	Minneapolis
Vance, John W.	Buffalo, N. Y.
Van Cleve, Horatio P.	Austin
Vanderpool, Thomas E.	Paynesville
Van Herik, Martin	Rochester
Van Meier, Henry	Stillwater
Van Rooy, George T.	Thief River Falls
Van Ryzin, Donald J.	Duluth
Varco, Richard L.	St. Paul
† Vastola, Anthony P., Jr.	Rochester
Vaughan, Victor M.	Truman
Vaughn, C. Gordon	St. Paul
Vaughn, Louis D.	Rochester
Veinbergs, Arnolds	Minneapolis
Veirs, Dean M.	St. Paul
Veirs, Ruby J. S.	St. Paul
Venables, Alexander E.	St. Paul
Vennes, Jack A.	Minneapolis
Veranth, Leonard A.	St. Cloud
Verby, John E., Jr.	Rochester
Vezina, John C.	Mapleton
Vik, A. Elliott	Minneapolis
Vik, Melvin	Cambridge
† Viren, Fred K.	Rochester
Virnig, Hildegard J.	Caledonia
Virnig, Mark P.	Wells
Virnig, Richard P.	Wells
Vitols, T. M.	Minneapolis
Vix, Vernon A.	Worthington
Vogel, Howard A. L.	New Ulm
Vollmer, Frederick J.	Winona
Von Drasek, Joseph	Mankato
Votel, Thomas W.	St. Paul

Waas, Charles W.	St. Paul
Wadsworth, George L.	Cambridge
† Wagener, Henry P.	Rochester
Wagner, Norman W.	Benson
Wagoner, James M.	Harmony
Wahlquist, Harold F.	Minneapolis
Wakai, Coolidge S.	Honolulu, Hawaii
Wakefield, Elmer G.	Rochester
† Wakim, Khalil G.	Rochester
Walder, Harold J.	Duluth
Waldmann, Edward B.	Rochester
Waldron, Carl W.	Hopkins
Walfred, Karl A.	St. Cloud
† Walker, A. E.	St. Paul
Walker, Arthur E.	St. Paul
Wall, Carl R.	Minneapolis
Wall, James O.	St. Paul
Wallace, Helen M.	Minneapolis
Wallace, Martin O.	Duluth
† Waller, Joseph D.	Pine City
Wallinga, Jack V.	St. Paul
Walonick, Albert L.	St. Louis Park
Walsh, Edward F.	St. Paul
Walsh, Francis M.	Minneapolis
Walsh, William T.	Minneapolis
Walter, Clarence W.	St. Paul
Walter, Frederick H.	International Falls
Walter, William E.	Wanamingo
Walters, Waltman	Rochester
Wandke, Otto E.	Fairmont
Wangensteen, Owen H.	Minneapolis
Ward, Louis E.	Rochester
Ward, Percy A.	Minneapolis
Warner, James J.	Perham
Warren, Cecil A.	St. Paul
† Warvel, John H., Jr.	Rochester
Wasmund, Clarence W.	Red Wing
Wasson, Loren F.	Alexandria
Waters, Alvin W.	Minneapolis
Watkins, Charles H.	Rochester
Watkins, Charles W.	Vancouver, Wash.
Watkins, John A.	Wells
Watson, Alexander M.	Royalton
Watson, C. Gordon	Minneapolis
Watson, Cecil J.	Minneapolis
Watson, John R.	Rochester
Watson, P. Theodore	St. Paul
† Watson, Percy T.	Miami, Fla.
Watson, Robert M.	Morris
Watson, Robert N.	Detroit Lakes
Watson, Sidney W.	Royalton
Watson, Virgil A.	Detroit Lakes
Watson, William H. A.	St. Paul
Watson, William J.	Newport
Wattenberg, Lee W.	Minneapolis
Watz, Clarence E.	St. Paul
Waugh, John M.	Rochester
Weatherhead, Donald S. P.	Minneapolis
¶ Weaver, Myron M.	Schenectady, N. Y.
Weaver, Paul H.	Faribault
† Weaver, W. F.	Rochester
Webb, Edgar A.	Minneapolis
* Webb, Roscoe C.	Minneapolis
Webber, Fred L.	St. Paul
Webber, Richard J.	St. Louis Park
Weber, Harry M.	Rochester
Weber, Lowell W.	Minneapolis
Wedes, Deno J.	St. Paul
Weed, Lyle A.	Rochester
Weeks, Richard E.	Rochester
Weidman, William H.	Rochester
† Weir, James F.	Rochester
Weir, Matthew J.	Virginia
Weis, Benjamin A.	St. Paul
Weisberg, Maurice	St. Paul
Weisberg, Rapheal J.	Minneapolis
Weiss, Carl A.	Hastings
Welch, John S.	Rochester
Wellman, William E.	Rochester
Wellner, Theodore O.	Rochester
Wells, Arthur H.	Duluth
Wells, Walter B.	Jackson
Wendland, John P.	Minneapolis
Wenner, Waldemar T.	St. Cloud
Wente, Harold A.	Rochester
† Wentworth, Albert J.	Mankato
Wenzel, Gilbert P.	St. Paul
Werner, George	Minneapolis
Wesolowski, Stanley P.	Minneapolis
† West, Catherine C.	Minneapolis
* Westby, Magnus	Madison
Westby, Norval M.	Madison
† Westerman, Alvin E.	Montgomery
† Westerman, Fred C.	Montgomery
Westley, Kent	Little Falls
Westover, D. E.	St. Paul
Westrup, John E.	Lanesboro
Wetherby, Macnider	Minneapolis
Wetteland, Thomas F.	W. St. Paul
Wetzel, Earl V.	St. Paul
Wexler, Harold M.	Minneapolis
Weyhrauch, William R.	Rochester
† Wheeler, Daniel W.	Duluth
Wheeler, Robert W.	Minneapolis
Whisnant, Jack Page	Rochester
Whitacre, John C., II	Minneapolis
Whitcomb, Fred F., Jr.	Rochester
White, Asher A.	Minneapolis
† White, James C.	Rochester
† White, S. Marx	Minneapolis
White, Willard D.	Minneapolis
Whitesell, Lloyd A.	Minneapolis
Whitson, Sidney A.	Albert Lea
Whittemore, Dexter D.	Bemidji
Wholey, Mark H.	Rochester
Widen, Wilford F.	Minneapolis
Wikoff, Howard M.	Crookston
† Wilbur, Dwight Locke, III	Rochester
Wilbur, Oscar M.	Hibbing
Wilcox, G. Charles	Albert Lea
† Wilder, Kenneth W.	Minneapolis
† Wilder, Robert L.	Minneapolis
† Wilder, Russell M.	Rochester
† Wilder, Russell M., Jr.	Topeka, Kansas
Wilder, Walter L.	Minneapolis
Wilken, Paul A.	Minneapolis
† Wilkowske, Rudolph J.	Owatonna
Will, Charles B.	International Falls
Will, W. W.	Bertha
*† Willcutt, Clarence E.	Phoenix, Arizona
† Williams, Arthur B.	St. Paul
Williams, Bruce F. P.	Duluth
† Williams, Charles A.	Pipestone
Williams, Clayton K.	St. Paul
‡ Williams, Francis R.	Duluth
Williams, George E.	St. Paul
Williams, Henry L., Jr.	Rochester
† Williams, Hugh O.	Lake Crystal
Williams, John A.	St. Paul
† Williams, Leon A.	Minneapolis
Williams, Mervyn M.	Ah-gwah-ching
Williams, Marland R.	Cannon Falls
Williams, Paul A.	Minneapolis
Williams, Richard A.	St. Paul Park
Williams, Richard E.	Robbinsdale
† Williams, Robert	Unknown
Williamson, Harold A.	Fairmont
Wilmer, Harry A.	Rochester
Wilmot, Cecil A.	Litchfield
Wilmot, Harold E.	Litchfield
† Wilson, Clyde E.	Blue Earth
Wilson, J. Allen	St. Paul
Wilson, James V.	St. Paul
Wilson, Louis J.	Winona
Wilson, Paul F.	Rochester
Wilson, Robert B.	Rochester
Wilson, Robert E.	Minneapolis
Wilson, Rolland H.	Winona
Wilson, Viktor O.	Rochester
Wilson, Warren E.	Northfield
Winchell, Paul	Minneapolis
Winge, H. C.	Wheaton
Winkelmann, Richard K.	Rochester
† Winnick, Joseph B.	St. Paul
† Winter, John A.	Duluth
‡ Winter, Malcolm D., Jr.	Ellsworth AFB, South Dak.
Winther, Nora M. C.	Minneapolis
Wipperman, Frederic F.	Minneapolis
† Wise, James K.	Rochester
† Witham, Carl A.	Minneapolis
Witter, Robert L.	Wadena
Witthaus, Melvyn E.	St. Paul
† Wittich, Frederick W.	Minneapolis
Wittrock, Louis H.	Watkins
Wohlrabe, Arthur A.	Minneapolis
Wohlrabe, A. Cabot	Minneapolis
Wohlrabe, Clarence F.	North Mankato
Wohlrabe, Edwin J.	Springfield
Wohlrabe, John C.	North Mankato
Wolf, Alfred H.	Minneapolis
* Wolff, Herman J.	St. Paul
† Wolff, John M.	Duluth
Wolkoff, H. J.	St. Paul
Wollaeger, Eric E.	Rochester
Wolstan, Simon D.	Minneota
Wolter, Frederick H.	Minneapolis
† Woltman, Henry W.	Rochester
*† Wood, Harry G.	Rochester
Wood, Lloyd T.	Forest Lake
† Woodington, George F.	Rochester
Woodruff, Whitney	Virginia
Woolner, Lewis B.	Rochester
Word, Harlan L.	St. Paul
Workman, Warner G.	Tracy
† Worlton, James T.	Rochester
Worrell, Janet	Rochester
† Woyda, William C.	Minneapolis
Wright, Robert R.	Austin
Wright, Thomas D.	Minneapolis
Wright, Wale S.	Minneapolis
Wright, William S.	Edina
† Wry, Paul E.	Rochester
* Wyatt, Oswald S	Minneapolis
Wynne, Herbert M. N.	Minneapolis

† Yaeger, Wilbert W.	Marshall
Ylvisaker, Ragnvald S.	Minneapolis
† Yoerg, Otto W.	Minneapolis
Yoss, Robert E.	Rochester
Young, Henry H.	Rochester
Young, Thomas O.	Duluth
Younger, Lewis I.	Winona
Youngren, Everett R.	St. Paul
Yue, Wen Y.	Oak Terrace

Zachman, Albert H.	Melrose
Zachman, Leo L.	St. Paul
Zagaria, James F.	St. Paul
Zahrendt, O. Lewis	Minneapolis
Zarling, V. Richard	Minneapolis
Zaworski, Leo A.	Minneapolis
* Zee, Urban H.	Mankato
‡ Zeller, Nicholas H.	Unknown
Zemke, Erhart E.	Fairmont
Zemmers, Roberts	Duluth
Zempel, Alan R.	Starbuck
Zierold, Arthur A.	Minneapolis
† Zimmer, Roy W., Jr.	Rochester
Zimmermann, Bernard	St. Paul
† Zimmermann, Harry B.	St. Paul
Zinn, Charles W.	Elk River
Zinter, Ferdinand A.	Minneapolis
Ziskin, Thomas	Minneapolis

Meetings and Announcements

NATIONAL

American Congress of Physical Medicine and Rehabilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

Gerontological Society, Inc., Bellevue Stratford Hotel, eleventh annual scientific meeting, Philadelphia, Pennsylvania, November 6, 7, and 8, 1958.

National Society for Crippled Children and Adults, 1958 convention, November 16-20, Statler Hotel, Dallas, Texas.

INTERNATIONAL

American College of Surgeons, sectional meeting, Stockholm, Sweden, July 2-7, 1958.

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Israel Medical Association, fourth world assembly, August 12-24, Tel Aviv, Jerusalem, and Haifa.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy, Association, Paris, France, October 19-26, 1958.

FRONTIERS OF CARDIOVASCULAR DISEASE

A one-day meeting devoted to the study of cardiovascular disease will be presented in Fergus Falls, June 28, 1958, by the Medical Staff of the Lake Region Hospital, Fergus Falls, Minnesota, in conjunction with the new Hospital Dedication.

Attendance at the meeting has been approved for seven hours' credit in Category I by the Academy of General Practice.

Program

Auditorium, Otter Tail Power Company
(125 South Mill Street)

Registration—8:30 A.M.

Morning Session—9:00 A.M.

"Venous Thrombosis and Pulmonary Emboli"—Ben Fuller, M.D., St. Paul, Minn.

"The Drugs We Will Use"—Ray Beiter, M.D., Professor of Pharmacology, University of Minnesota

"Occlusive Arteriosclerosis"—Morley Cohen, M.D., Winnipeg, Canada

Coffee—10:30 A.M.

"Abdominal Aneurysms"—Richard Varco, M.D., Professor of Surgery, University of Minnesota

Panel Discussion—11:15 A.M.

"Anticoagulants in the Management of Cardiovascular Disease"

Luncheon—12:15 P.M.

River Inn Hotel
Courtesy Fergus Falls Medical Staff

Hospital Dedication Ceremony—1:30 P.M.
(On Hospital Grounds)

Afternoon Session—2:00 P.M.

"Fat, Diet and Atherosclerosis"—Ivan Frantz, M.D., Professor, Department of Medicine, University of Minnesota

"Essential Hypertension"—E. G. Brownell, M.D., Winnipeg, Canada

"Vascular Aspects in Toxemia of Pregnancy"—Arthur Hunt, M.D., Mayo Clinic, Rochester

"Cerebrovascular Syndromes"—Clark Millikin, M.D., Mayo Clinic, Rochester

Coffee—4:00 P.M.

"Angiocardiography and Cineradiography in the Diseases of the Circulatory System"—Richard Lester, M.D., Assistant Professor of Radiology, University of Minnesota

Discussion—"Open Heart Surgery"
"Congenital Lesions"—Morley Cohen, M.D.
"Acquired Lesions"—Richard Varco, M.D.

Social Hour and Outdoor Buffet—6:30 P.M.
For doctors and wives at a beautiful Minnesota lake
Introduction of guests

Women's Program

Registration—9:00 A.M.
Lake Region Hospital
Main Entrance
Hospital Tour

Coffee—10:30 A.M.
Hospital Cafeteria

Luncheon—12:15 P.M.
Ranch House Restaurant
Courtesy Medical Staff

Hospital Dedication Ceremony—1:30 P.M.
Social Hour and Banquet—6:30 P.M.

Other Functions

June 20, 1958

Hospital Benefit Ball—9:00 P.M.
Under auspices of Junior Chamber of Commerce
Fergus Falls Armory

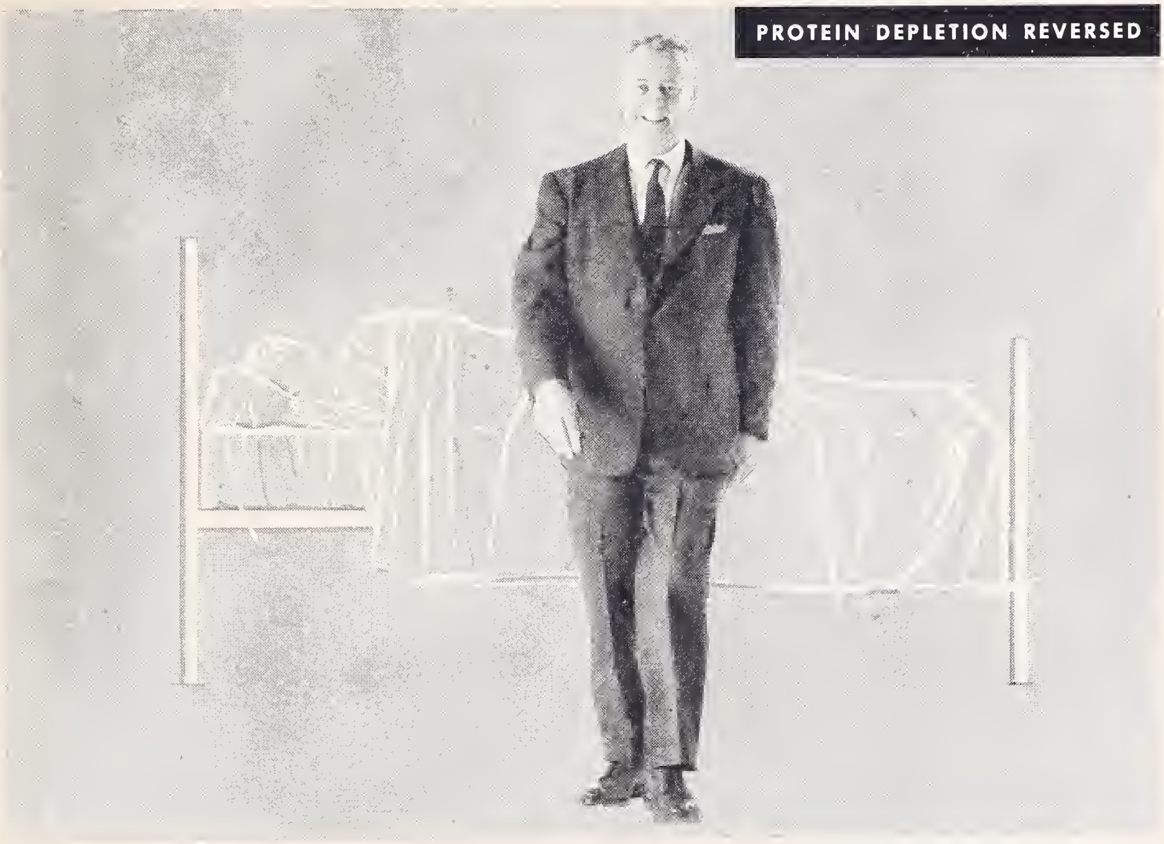
June 21, 1958

Public Meeting—8:00 P.M.
Hospital Dedication
Speech—Dr. Walter Judd, U. S. Representative
Roosevelt Park High School

ANESTHESIA MEMORIAL FOUNDATION RULED TAX-EXEMPT AND CONTRIBUTIONS TAX-DEDUCTIBLE

The Anesthesia Memorial Foundation, a national organization established in 1956 primarily to provide grants-in-aid to physicians for graduate training in anesthesiology, on April 21, 1958, was given the status of "an organization operating exclusively for charitable and educational purposes" by the United States Treasury

(Continued on Page A-42)



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- **The patient recovers faster**

Similarly Nilevar helps correct the "protein catabolic state" associated with prolonged bed rest in carcinomatosis, tuberculosis, anorexia nervosa and other chronic wasting diseases.

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ANESTHESIA MEMORIAL FOUNDATION

(Continued from Page 456)

Department, according to Mr. John Lansdale, Jr., of Cleveland, Ohio, secretary of the foundation. The effect of this ruling, Mr. Lansdale said, is that the foundation itself is exempt from federal income taxes and gifts to the foundation are deductible from the federal income taxes of the donors.

The objective of the Anesthesia Memorial Foundation is to provide financial aid for physicians who plan to specialize in anesthesiology but who lack the funds with which to carry out graduate training. The foundation will advance recipients of the grants-in-aid up to $1,000 a year for the specified purpose, and the current program anticipates the expenditure of approximately $500,000 for grants-in-aid before the money lent for aid begins to be returned.

COLLOQUY ON ADVANCES IN MEDICINE

The second Oklahoma Colloquy on Advances in Medicine will be held on November 12, 13, 14 and 15, 1958. It will be devoted to Arthritis and Related Disorders and is under the joint sponsorship of the Department of Medicine, University of Oklahoma, the Division of Postgraduate Education.

BIRTH RECORDS ENTERED ACCORDING TO CENTRAL DAYLIGHT TIME

The Minnesota Department of Health reminds physicians that births or deaths occurring during the pendency of what is commonly known as "Central Daylight Time" should be recorded pursuant to that time. The following is from the office of Governor Freeman, April 24, 1958: Central Time begins 2 a.m., April 27, at which time clocks will be advanced to 3 a.m. Central Daylight Time ends 2 a.m., September 2, at which time clocks will be set back to 1 a.m. It might be well to add the abbreviation CDT after the time on birth and death certificates.

MEDICAL SCHOOL ADMISSIONS TO BE INCREASED

Admissions to the University of Minnesota Medical School will be increased from 125 to 140 students for the next school year (1958-59) and to 150 in 1959, Dr. Robert B. Howard, associate dean of the College of Medical Sciences, has announced.

By 1962, the four medical school classes will have a combined total enrollment of approximately 600 as compared with under 500 at present.

Enlarged and improved laboratory facilities and strengthening of the staffs of the University's basic medical sciences departments are the principal factors which permit the step-up in enrollment, Dr. Howard explained. The basic medical sciences include: anatomy, pathology, physiological chemistry, pharmacology and bacteriology.

"This move will ultimately produce more physicians to care for the people of our state," Dr. Howard stated.

About 300 applicants now seek admission to the medical school each year, according to Dr. Howard. A minimum of three years of college is required for entrance, and the medical school course leading to a doctor of medicine degree takes four years to complete.

In announcing the upping of the admissions limit, Dr. Howard emphasized that the University Medical School has absolutely no admissions quotas based on race, sex, religion or father's occupation—"contrary to rumors that such quotas exist."

MINNESOTA SURGICAL SOCIETY

At the recent meeting of the Minnesota Surgical Society in Rochester, the following were elected to positions in the Society: President, Dr. Arnold J. Kremen, Minneapolis; Vice-President, Dr. Donald E. Stewart, Crookston, and Secretary-Treasurer, Dr. Oliver H. Beahrs, Rochester. The directors are Dr. Owen G. McDonald, Duluth; Dr. P. W. Harrison, Worthington; Dr. Charles E. Rea, Saint Paul, and Dr. David P. Anderson, Austin.

The next three meetings of the Society are to be held as follows: November 7, 1958, Saint Paul; July 17, 1959, Duluth; October 30, 1959, Minneapolis.

CLINICAL-PATHOLOGICAL CONFERENCE

(Continued from Page 406)

cases showed simple destruction of liver parenchyma by tumor. Incidentally, these same five cases represented 22 per cent of our twenty-three cases of common bile duct obstruction from any cause, including calculi, postoperative structure and malignancy.

Dr. Bell: I would like to ask Dr. Horns to comment on the negative cephalin flocculation in the presence of an elevated bromsulphalein retention and a huge liver filled with metastases.

Dr. Horns: One can see considerable liver destruction and even cirrhosis with a normal cephalin cholesterol. I recall one patient who died in hepatic coma, with a normal cephalin cholesterol. The cephalin cholesterol test reflects an abnormal albumin. The thymol turbidity is thought to reflect an abnormal globulin. The cephalin cholesterol test is said to be more likely elevated in cirrhosis associated with alcoholism. The thymol turbidity is said to be more likely elevated in postnecrotic cirrhosis and may reflect a prolongation of hepatitis.

Dr. Bell: Is the bromsulphalein retention one of our best tests of liver function?

Dr. Horns: It is a very sensitive test and may give more false positive results than others do.

Final Anatomic Diagnoses

1. Malignant cholangioma of liver with metastases to the lungs, right adrenal and regional lymph nodes.
2. Chronic lymphocytic leukemia, aleukemic phase.

References

1. Gross, L.: Acta haemat., 13:13, 1955.
2. Ewing, J.: Neoplastic Diseases, 4th ed., pp. 738-752. Philadelphia: W. B. Saunders Co., 1940.

Woman's Auxiliary

MOWER COUNTY AUXILIARY REVIEWS TWENTY-SEVEN-YEAR HISTORY

In February, 1931, ten physicians' wives met and organized the Mower County Medical Society Auxiliary. Until ten years ago, the average attendance was nine. These few women accomplished miracles! From 1931-1935, they made layettes by the dozens. In 1934, they gave twelve complete layettes besides many assorted individual items. A typical layette was sent to the State Medical Association meeting for an exhibit and in 1935 one was sent to the American Medical Association Convention. Every Christmas, baskets of food and toys were given to the needy.

These ambitious auxiliary members found time to fold many of the surgical dressings for the hospital. They gave a card party, sold over 200 tickets at 50 cents apiece, secured donations, and emerged with a profit of $213.94. Mrs. J. K. McKenna's expenses were $1.35—seems a bit high, doesn't it? With some of this money, they bought an oxygen tent for the hospital. They also bought a rubber runner for the hospital corridor, costing $20.00.

In 1936, another card party netted only $60.00. With this amount, a surgical spotlight was purchased. Hospital Day in 1937 found the doctors' wives ably assisting at that event. In 1938, a gift of $20.00 was given to the hospital.

A much-needed new addition to St. Olaf Hospital, Austin, was opened in 1940. Auxiliary members sewed 260 pairs of drapes for the rooms. They also served coffee and tea for the opening. The group started sewing for the Red Cross in 1941. In 1943, they bought a canopy for the oxygen tent and twelve new bedspreads. Members sold War Savings Stamps in 1944. Forty-one subscriptions to THE BULLETIN were sold in the state, and of these eight were sold in Mower County. A rummage sale netted $58.36 with which money an electric cautery was purchased.

Cancer dressings were made during the monthly meetings and, in 1945, the group purchased a Lycos Sphygmomonometer.

Auxiliary members assisted with the Heart Fund Drive in 1948 by placing plastic hearts for contributions in business places and collecting them at the close of the drive.

The year 1951 marked the beginning of a series of "teas" given for high school girls in the attempt to interest them in nursing and fields allied to medicine. A definite shortage has developed and physicians' wives are in a key position to do something about it. The Auxiliary purchased a $100.00 suction apparatus for the hospital during this year. Mrs. C. L. Sheedy was named president-elect of the State Auxiliary in 1951, and Mrs. B. J. Cronwell was named as delegate to the AMA Auxiliary convention. Members collected sample drugs from physicians' offices, sorted and packed them and

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Phenobarbital	16.2 mg. (¼ gr.)
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Reserpine	0.1 mg.
Thiamine hydrochloride	5.0 mg.

SUPPLIED: Bottles of 100 scored tablets.

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sent them to missionary fields. Favors were made for the hospital trays for Christmas.

Mrs. C. L. Sheedy brought honor and distinction to Mower County by being elected and serving as president of the State Auxiliary in 1952-53. That same year, Mower County won third places in *Hygeia* sales, with Mrs. Tracy Barber as chairman.

In 1952 members took part in the National Smoking Survey conducted to determine the incidence of cancer as related to smokers and non-smokers.

A Public Relations Fund was proposed in 1953, and since that time members contribute voluntarily toward it, contributions being sent to the AMEF Fund. Recruitment teas have continued each year with a growing interest. Programs on mental health, legislation, civil defense and safety, with member participation, are held each year. Mrs. R. VanCleve served as state *Today's Health* chairman for two years, Mrs. S. Peterson served as state recruitment chairman in 1955-56 and as state recording secretary in 1957-58.

Along with progress noted in this centennial year in Minnesota, Mower County members wish to make note of their progress as a Medical Society Auxiliary, from ten members in 1931 to thirty-three at present. Meetings are held in the homes with good attendance and enthusiastic participation.

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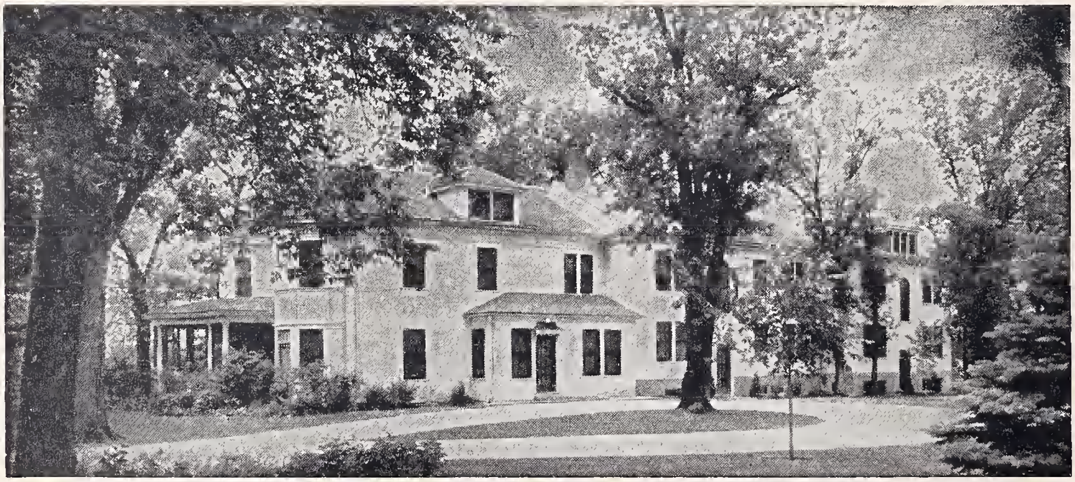

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In Memoriam

ORRIMAN S. ELY

Dr. Orriman S. Ely, South Saint Paul city health officer for forty years, died April 29, 1958, in Miller Hospital, Saint Paul. He was seventy-nine years of age. In addition to his duties as city health officer, Dr. Ely had practiced medicine in South Saint Paul since 1916.

Born in Middletown, Ohio, he moved to Wisconsin and was graduated from the Superior Normal school in 1900. He was principal of the Chetek High School two years.

He was graduated from the University of Minnesota medical school in 1906. After serving his internship in St. Joseph's Hospital, Saint Paul, he practiced medicine eight years at Wallace, South Dakota.

He was a member of Luther Memorial Church, the Ramsey County Medical Society, a life member of the Minnesota State Medical Association and the American Medical Society.

Surviving are his wife, the former Claire Kittelson of Henry, South Dakota, and four children, Mrs. E. W. (Dorothy) Horst and Mrs. Elmer (Barbara) Bester, both of South Saint Paul; Dr. R. S. Ely of Salt Lake City, Utah, and Rev. Albert Ely of Paris, France. Another son, William, died while serving in the armed forces in 1942. Ten grandchildren also survive.

ROWLAND GILMORE

Dr. Rowland Gilmore, former Bemidji surgeon, died April 14, 1958. He was eighty-nine years old. Dr. Gilmore was granted an affiliate membership in the Minnesota State Medical Association in 1938. He was also a member of the Upper Mississippi Valley Medical Association and a member of the American College of Surgeons.

FRANK J. LEXA

Dr. Frank J. Lexa, prominent Lonsdale physician and civic leader, died April 11, 1958. He was seventy-five years old.

Dr. Lexa was found dead at his home in Lonsdale on Thursday evening, April 17. He had planned to leave on Saturday, April 12, to visit his son, Francis, in Chicago. When he failed to arrive, his son called Lonsdale authorities and Policeman Joseph Pumper and Mayor Robert Novak went to the Lexa home where the physician was found dead.

Dr. Neutzman, Rice County coroner, ruled that death was from natural causes. It is believed that Dr. Lexa had been dead since Friday, April 11.

Dr. Lexa was born in Wheatland township on December 6, 1882, the son of the late Mike and Anna Vita Lexa. He received his early education at the Iowa City Academy and received his Doctor of Medicine degree from the University of Illinois in 1906. He served his internship at Augustine Hospital in Chicago and shortly thereafter started medical practice at Lonsdale.

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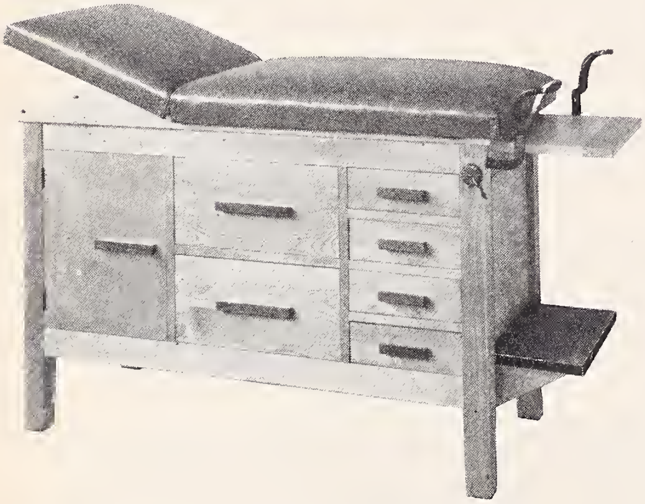

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This sturdily constructed table is made of the finest genuine Canadian Birch—solid wood legs and rails with birch-face five-ply panels for drawer and door fronts. It is offered in one standard finish only—natural wood in a golden blond color. It has six drawers and one compartment accessible from either side. There is a three position headrest, sliding stirrups, sliding leg rest and foot step. Upholstered with brown DuPont Fabrilite with 2" of padding.

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Length 48", fully extended 68", width 22" height 32".

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He was married to Anastacia R. Remes at Lonsdale in November, 1916.

He is survived by his wife; a son, Frank J. Lexa, Jr., an attorney-at-law in Chicago, Illinois; two daughters, Mrs. Larry (Helen) Johnson of Windom and Miss Alice Lexa of West Palm Beach, Florida; four grandchildren; two brothers, Mike of Lonsdale and Dr. William G. of Chicago; and two sisters, Mrs. Emma Wondra of New Orleans, Louisiana, and Mrs. Ida Benzik of Leavenworth, Kansas.

Dr. Lexa was a member of the Faribault Elks Club and the Minneapolis Athletic Club.

He served as president of the State Bank of Lonsdale from 1930 until 1948.

He was a member of the Rice County Medical Society and was granted a life membership in the Minnesota State Medical Association in May, 1953. In 1956, he was awarded citations by the American Medical Association, the Minnesota State Medical Association and the Rice County Medical Society for fifty years of service.

He served for many years as health officer for the village of Lonsdale and was physician and surgeon for the Chicago, Milwaukee and Saint Paul Railroad.

He was a prominent leader in the Republican party in this area and held numerous party offices, including chairmanship of the Rice County Republican party.

Dr. Lexa was a member of the Association of Railroad Surgeons, a staff member at St. Lucas Hospital at Faribault, and the Independent Bankers Association.

ARCHIBALD H. LOGAN

Dr. Archibald H. Logan, a member of the staff of the Mayo Clinic from 1909 to 1946 and head of a section in medicine for most of that time, died May 1, 1958, at St. Mary's Hospital of a cardiovascular disease. He was eighty years old and had been in the hospital since December after an illness of three years.

For four years, from 1925 to 1929, Dr. Logan served as faculty chairman of the Clinic, a title currently known as president of the staff.

Dr. Logan was born June 25, 1877, at Pittsburgh, Pennsylvania. From 1896 to 1898, he was a student at Washington and Jefferson College, Washington, Pennsylvania, but his education was interrupted by service in the Spanish-American War, in which he served as a private and corporal.

He received the degree of Doctor of Medicine in 1907 from the University of Pennsylvania. From July, 1907 to July, 1908, he was an intern in Mercy Hospital in Pittsburgh, and in 1908-1909 he was an instructor in medicine in the University of Pittsburgh. Also he carried on the private practice of medicine in Pittsburgh.

Dr. Logan came to Rochester as an assistant in medicine in the Mayo Clinic, December 31, 1909. On January 1, 1911, he was appointed head of a section in medicine, a post he occupied with distinction until he became a senior consultant in 1942. He retired on

April 1, 1946. He opened the fourth diagnostic office to be established at the Mayo Clinic.

Washington and Jefferson College conferred the honorary degree of Master of Science upon Dr. Logan in 1930, and in 1934 awarded him the honorary degree of Doctor of Science. In 1938, he was certified as a specialist in internal medicine by the American Board of Internal Medicine, and the same year was elected president of the Rochester Chapter of Alumni of the University of Pennsylvania. In 1940, in association with colleagues at the Mayo Clinic, Dr. Logan was awarded the silver medal of the American Medical Association for an exhibit on polyps of the rectum and colon.

Dr. Logan was appointed an assistant professor of medicine when the Mayo Foundation was created as a part of the Graduate School of the University of Minnesota in 1915. He was promoted to associate professor in 1918. He was a member of the Minnesota State Medical Association, American Medical Association, and the Alumni Association of the Mayo Foundation.

Dr. Logan was active in the affairs of the Presbyterian Church from the time he came to Rochester as a young man. He was superintendent of the Sunday school for many years and was also a member of the sessions of the church.

He married the former Amy H. Dunlap of Washington, Pennsylvania, on August 6, 1908, at Muskoka, Ontario, Canada. She survives with four children: Dr. George B. Logan, a member of the pediatrics staff at the Mayo Clinic; Mrs. John M. Waugh, Rochester; Dr. Archibald H. Logan, Jr., Spokane, Washington, and John T. Logan, Chappaqua, New York, thirteen grandchildren, and a sister, Miss Henrietta B. Logan of New York City.

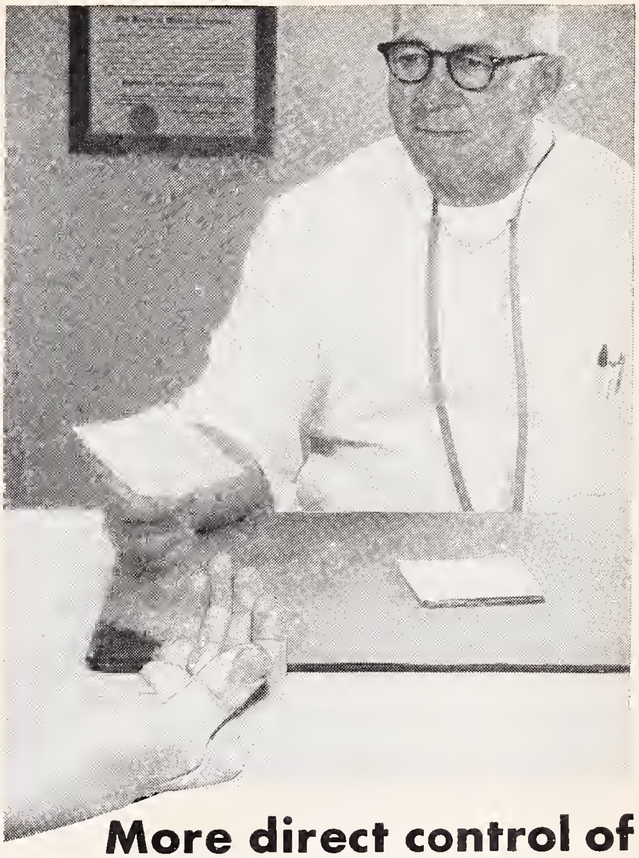
ROBERT DANIEL MUSSEY

Dr. Robert Daniel Mussey, a member of the staff of the Mayo Clinic for forty years, died in St. Mary's Hospital in Rochester on April 20, 1958.

Dr. Mussey was born in Glendale, Ohio, on January 10, 1884. He received the degree of Doctor of Medicine from the University of Cincinnati in 1908; in 1908 and 1909 he was an intern in the Cincinnati General Hospital, and in 1909 and 1910 he was receiving physician in that hospital.

Dr. Mussey came to Rochester, Minnesota, on July 1, 1910, as an intern in Saint Mary's Hospital. On April 1, 1916, he was named head of a section of medicine. From 1917 to 1919, Dr. Mussey served as a captain and then major in the Medical Corps of the U. S. Army. He was assistant professor of medicine in the Mayo Foundation, Graduate School, University of Minnesota, from 1915 to 1921 and associate professor of medicine in 1921 and 1922.

On October 1, 1921, he was appointed head of the Section of Obstetrics and Gynecology, a post he occupied until July 1, 1947, when he became chairman of



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two sections on obstetrics and gynecology. On January 1, 1949, he became a senior consultant. He also served as associate professor of obstetrics and gynecology in the Mayo Foundation, Graduate School, University of Minnesota, from 1922 until 1928, and was professor from 1928 until his retirement from the Mayo Clinic and Mayo Foundation on March 31, 1950.

He was president of the Minnesota Society of Obstetrics and Gynecology in 1937, of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons in 1948, and of the Central Association of Obstetricians and Gynecologists in 1938. In 1937, he was chairman of the Minnesota Committee on Maternal Welfare, and he has been a director of the American Committee on Maternal Welfare.

Dr. Mussey also was a member of the American Medical Association, American Gynecological Society, the Minnesota Academy of Medicine, an associate member of the Minnesota State Medical Association, the Society of the Sigma Xi, Alpha Omega Alpha medical honor society and the Alumni Association of the Mayo Foundation.

Dr. and Mrs. Mussey had five children: Dr. Mary Elizabeth Mussey, a member of the Section of Obstetrics and Gynecology of the Mayo Clinic; Frances Gano Mussey (Mrs. David T. Jennings of Williamsport, Pennsylvania); Dr. Robert D. Mussey of Urbana, Illinois; Madge Ayres Mussey (Mrs. Paul J. Bilka of Excelsior, Minnesota); and Dr. William Clive Mussey of Madison, Wisconsin.

A sister, Mrs. C. S. Pinkerton, lives at Wauwatosa, Wisconsin.

JOHN R. MEADE

Dr. John R. Meade, prominent Saint Paul physician, died May 4, 1958, at his home. He was fifty-five years of age. Death was due to a heart ailment.

He was a native of Saint Paul and a graduate of St. Thomas College and the University of Minnesota medical school, class of 1930. Following graduation, he spent three years on the Mayo Clinic staff in Rochester, and was the first Mayo Clinic Fellow to return to private practice in Saint Paul.

He set up the first diabetes clinic at Ancker Hospital in about 1935. He was on the teaching staff of the University of Minnesota medical school and was active on the staffs of Miller, St. Joseph's and Ancker Hospitals.

A specialist in internal medicine, he was certified by the American Board of Internal Medicine. He was a member of the Ramsey County Medical Society, the Minnesota State Medical Association, the American Medical Association, the American Diabetes Association and the Medical Forum. He was a past president of the University Club.

He is survived by his wife, Lucille; a brother, Harry C., Minneapolis, and a sister, Mrs. Elizabeth Henry, Stillwater.

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Minnesota State Board of Medical Examiners

LICENSE OF MINNEAPOLIS PHYSICIAN SUSPENDED FOR FIVE YEARS

On May 10, 1958, the Minnesota State Board of Medical Examiners suspended the license to practice medicine and surgery held by Dr. James E. Fearing, thirty-seven, 5018 Emerson Avenue South, Minneapolis, for a period of five years. Dr. Fearing had been charged in a citation issued by the Board on June 20, 1957, with habitually indulging in the use of drugs, namely, desoxyn hydrochloride and dexedrine, and with having been convicted on or about May 4, 1957, of the offense of disorderly conduct in the village of Spring Lake Park, Minnesota. The citation also contained a review of the history of Dr. Fearing's case going back to March, 1949, when he was first ordered to appear before the Board in connection with his personal use of narcotic drugs and barbiturates. Because he had been undergoing hospitalization under the care of a physician, his case was continued by the Board on several occasions to the meeting of the Board on May 10.

Dr. Fearing's medical license was previously suspended by the Board for a period of three years on May 15, 1952, because of his personal use of narcotic drugs. In the fall of 1956, he again resumed the practice of medicine at Aitkin, Minnesota. Dr. Fearing, who was born in Crosby, Minnesota, on April 17, 1921, attended the medical school of the University of Minnesota where he received an M.D. degree in 1946. He had been licensed in Minnesota by examination in 1945 after obtaining his M.D. degree and thereafter he served a one-year internship in Harper Hospital, Detroit, Michigan. He began the practice of medicine in 1947 in Pequot Lakes, Minnesota, where he remained until March, 1949. Since that time, Dr. Fearing has also done some institutional medical work.

TWO QUACKS CONVICTED BY JURY AT LITTLE FALLS

On May 5, 1958, H. H. Unruh and Noah Gering were convicted by a jury in Little Falls, Minnesota, of the crime of maintaining an office for the practice of healing without first obtaining a certificate of registration in the basic sciences. The Hon. Byron R. Wilson, Judge of the District Court of Morrison County, thereupon sentenced each defendant to one year in the Morrison County jail and then stayed the execution of the sentences on the condition that the defendants refrain from the practice of healing in Minnesota and that they pay the costs of their prosecution. Judge Wilson ordered that both defendants be placed under the supervision of the State Board of Parole in Minnesota.

After an investigation by representatives of the Minnesota State Board of Medical Examiners disclosed that Unruh and Gering had an office in their home on Lake Alexander in Morrison County where they were treating numerous patients for such diseases as cancer, diabetes and arthritis, a complaint was issued on March 13, 1958, by Mr. Attell P. Felix, Morrison County attorney, charging the defendants with violating the basic science law as referred to above. An examination of the reception book kept by Unruh and Gering in their office revealed that, in a period of approximately four months, they had treated 720 patients for each of which treatments they admitted receiving a fee of from $4 to $5. Upon being questioned at the time of their arrest, the defendants described their system of healing as "wireless anatomy" and "polarity therapy." Several machines designated as a "Ray and Radiation Instrument," an "Oscillator" and a "Vibrosage" were found in the defendants' treatment rooms, but both Unruh and Gering admitted that each patient was given a vigorous rubbing on the bottom of the feet with "Unruh's Ointment."

At their arraignment in District Court on April 14, 1958, before Judge Wilson, both defendants pleaded not guilty. On May 2, 1958, trial of both defendants began at Little Falls. Testimony by the state's witnesses showed that neither defendant had a basic science certificate and that they were treating patients at their home for various ailments and making a charge of at least $4.00 for each treatment. The defendants each testified in their own behalf and admitted on cross examination that they had treated people for a fee. Unruh said he was forty-four years of age and that he had moved to Lake Alexander on November 25, 1957, from Monroe, South Dakota. He stated that he had been a farmer until six years ago when he started practicing "wireless anatomy." Gering said he was fifty-five, married and the father of four children. He also stated he was born in Kansas and had been a farmer all his life. According to Gering's testimony, he came to Minnesota from Harrold, South Dakota. Neither of the defendants has had any training in medicine or any other field of healing, nor does either hold a license to practice any form of healing in Minnesota.

HEALER SENTENCED IN ANOKA COUNTY

The defendant, Hugh Henderson, appeared on April 7, 1958, before the Hon. Leonard Keyes, Judge of the District Court of Anoka County at which time he pled guilty to the charge of a violation of the basic science act in that he maintained an office for the practice of healing without a basic science certificate. Judge Keyes sentenced Henderson to a term of four months in the workhouse of the City of Minneapolis. However, the judge stayed the execution of the sentence for a period of one year and placed the defendant on probation to the Minnesota State Board of Parole on the condition that he refrain from practicing any form of healing.

Henderson was arrested on January 22, 1958, at his home at 9500 Dunlap Ave. N. in the village of Lexington, Anoka County following an investigation by an inspector of the Federal Pure Food and Drug Administration and a representative of the Minnesota State Board of Medical Examiners. The investigation revealed that the defendant treated patients and made a charge of $2.00 for his services. Henderson's form of treatment was manual and vibrator massage and an adjustment of the neck and spine. The defendant also had vitamins, diuretic stimulants, nasal spray, laxative tablets, food supplements and vitamins for sale in his waiting room, all in violation of law.

A complaint was issued by the Anoka County Attorney's office in which Henderson was charged with the basic science violation. The defendant waived a preliminary hearing before Judge James Knutson of the Municipal Court of Anoka and was bound over by Judge Knutson to the District Court of Anoka County with the result as stated above.

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(1) Hagedorn, A. B.: Proc. Staff Meet. Mayo Clin. 32:705 (Dec. 11) 1957.
(2) Best, W. R.; Louis, J., and Limarzi, L. R.: M. Clin. North America (Jan.) 1958, p. 3.

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General Interest

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* * *

Dr. Gordon R. Kamman, Saint Paul, and Mr. Charles Murnane, Saint Paul attorney, addressed a joint meeting of the Beltrami County Medical Society and the district Bar Association at Bemidji, Minnesota, on Monday, May 5, 1958. Their topic was "Medico-legal Problems."

* * *

The May regular meeting of the **Minnesota Society of Neurology and Psychiatry** was held at the Mayo Clinic, Rochester, Minnesota, Saturday, May 31.

* * *

Dr. Adelaide M. Johnson, Rochester, clinical professor of psychiatry in the University of Minnesota, addressed members of the staff and fellows of the Los Angeles Psychoanalytic Institute on April 14. Dr. Johnson spoke on "New Techniques in the Management of Certain Personality Disorders."

* * *

American Academy of General Practice officers elected at the recent Tenth Annual Assembly in Dallas include two Minnesotans: vice president, **Dr. Charles C. Cooper**, and treasurer, **Dr. Albert E. Ritt**, both of Saint Paul.

* * *

St. Francis Hospital, La Crosse, Wisconsin, has announced that **Dr. Philip H. Utz**, who opened offices in La Crescent last November, is one of the newly appointed physicians to the St. Francis Hospital medical staff.

* * *

A special visit to the U. S. Naval Hospital at Great Lakes, Illinois, by **Dr. John S. Lundy**, of the Section of Anesthesiology of the Mayo Clinic, is the subject of a special feature of the April issue of *The Caduceus*, monthly publication of the Great Lakes Naval Hospital.

Dr. Lundy spent the morning of March 14 in the operating rooms of the hospital, demonstrating and teaching advanced techniques to Navy anesthesiologists and to personnel from the Downey Veterans Administration Hospital. In the afternoon, he lectured on current principles in the physiologic, mechanical and therapeutic effects of various agents and procedures.

* * *

Sunday, April 27, was Dr. Patterson Day in Fergus Falls. Residents of the county and the state joined with Fergus Falls citizens in recognizing the contribution made by **Dr. W. L. Patterson**, superintendent of the Fergus Falls State Hospital, to the mental health program of the state. He has been a member of the hospital staff for forty-six years, serving as superintendent for the greater part of that time.

Physicians of the Breckenridge Clinic have been in their new quarters at 227 North Sixth since Monday, May 5. The new Breckenridge Clinic is in full operation with a staff of five doctors. An open house to acquaint the public with the beautiful new quarters was held on Saturday, May 10.

* * *

Dr. E. N. Milhaupt, St. Cloud nose specialist, participated in an extensive postgraduate course in reconstructive nasal surgery presented at the University of Cincinnati College of Medicine and Cincinnati General Hospital, April 12-19.

* * *

An expectant mothers' course is being co-sponsored by the Mower County Medical Society and the County Public Health Nursing Service. The course started April 25 and will continue through June 4.

* * *

Appointment of **Dr. Winchell McK. Craig** as Rochester civil defense director has been announced by Mayor Alex P. Smekta.

* * *

Dr. Donald B. Peterson, superintendent of Anoka State Hospital, will serve also as acting superintendent of Hastings State Hospital until the new superintendent takes over on August 1.

* * *

Members of the Minnesota Obstetrical and Gynecological Society gathered recently in Rochester for the group's 1958 spring meeting. **Dr. E. A. Banner** of the Mayo Clinic is serving his fourth term as secretary-treasurer of the group. Other officers are **Dr. John L. McKelvey** of the University of Minnesota School of Medicine, president, and **Dr. Eugene M. Kasper** of Saint Paul, vice president.

* * *

Dr. Frank H. Krusen, head of the Section of Physical Medicine and Rehabilitation of the Mayo Clinic, has been appointed a member of the National Advisory Council on Vocational Rehabilitation by U. S. Secretary of Health, Education and Welfare.

* * *

Dr. Donald V. Smith, Blue Earth physician and surgeon for the past eight years, will leave his practice in July and move with his family to Seattle, Washington.

* * *

Award of a $42,840 cancer research grant to **Dr. Jerome T. Syverton** of the University of Minnesota School of Medicine has been announced by Stanley Hawks, president of the Minnesota division of the American Cancer Society.

The funds, part of a five-year continuing grant, will be used for support of a broad program in research in cells, viruses and cancer by the professor and head of the University's department of bacteriology and immunology.

(Continued on Page A-56)

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(Continued from Page A-54)

Participating in the project are scientists trained in such fields as immunology, virology, genetics, physiology, pathology, cytology and immunochemistry.

* * *

Dr. A. H. McFarland was elected president of the board of trustees of Franklin Hospital Association at the annual meeting. Other officers are **Dr. Wayne S. Hagen,** vice president; H. A. Blomgren, secretary, and A. G. Stasel, treasurer. Elected trustees were Stanley S. Gillam, **Dr. L. K. Buzzelle** and Winthrop M. Wadsworth.

* * *

Dr. John G. Minder, formerly of Alma, Nebraska, has joined the staff of **Drs. Kenneth Romness** and **Charles V. Carlson** in Mound.

* * *

Dr. D. C. Dahlin, of the Mayo Clinic surgical pathology section, recently presented a paper on "Neoplasma of Bone" at the joint meetings of the International Academy of Pathology and American Association of Pathologists and Bacteriologists held in Cleveland April 21-26. About 1,200 pathologists from throughout the country attended.

* * *

Dr. Arnold J. Kremen, Minneapolis, has been elected president of the Minnesota Surgical Society, it was announced Sunday, May 11. Other new officers are **Dr. Donald Stewart,** Crookston, vice president, and **Dr. O. H. Beahrs,** Rochester, secretary-treasurer.

* * *

Dr. Leonard S. Arling of the Northwest Industrial Clinic, Minneapolis, has been named secretary of the Industrial Medical Association at the current National Industrial Health conference, in Atlantic City, New Jersey.

* * *

The Royal College of Surgeons of England has named **Dr. Charles W. Mayo** an honorary fellow, a distinction given to only a small number of American surgeons throughout the years.

Only other Minnesotans or former state residents to receive this honor, as far as can be determined, were Dr. Mayo's father, Dr. Charles H. Mayo, in 1920; his uncle, Dr. William J. Mayo, in 1913; **Dr. Donald C. Balfour** of Rochester, retired Mayo Foundation director, in 1939; the late Dr. Fred Rankin of Lexington, Kentucky, brother-in-law of Dr. C. W. Mayo and former head of a section of general surgery at the Clinic, who left Rochester in 1933 and was named a Royal College fellow in 1953, and the late Sir Henry Wellcome of London, who lived at Garden City, Minnesota, and Rochester at one time before moving overseas. He was elected to the college in 1932.

Dr. Charles W. Mayo and his wife will go to London in July for the official conferring ceremony. They will leave Rochester June 27 and return July 14.

* * *

Dr. John F. Briggs, Saint Paul internist, was recently honored with a distinguished service award by the Minnesota Heart Association's board of directors. Dr.

(Continued on Page A-58)

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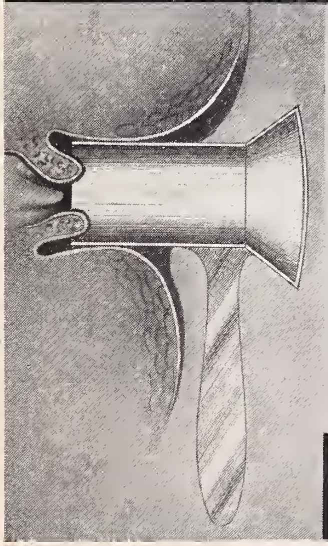

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(Continued from Page A-56)

Briggs helped found the association in 1948 and served as its president in 1950. He has practiced medicine in Saint Paul for thirty years. As chairman of the Minnesota Heart Association's professional education committee he spoke recently on "Surgery in Heart Disease" at a meeting of the Student American Medical Association in Chicago.

The directors also named **Dr. Helen M. Wallace**, Minneapolis, chairman of a newly created committee on school health. She is professor of maternal and child health at the school of public health, University of Minnesota.

* * *

Dr. James H. DeWeerd, Mayo Clinic urologic surgeon, has been named first vice president of the newly organized Paul Watkins Memorial Methodist Home at Winona.

* * *

Dr. Howard F. Polley, consultant in medicine at the Mayo Clinic since 1943, is one of two prominent alumni of Ohio State University who received Alumni Achievement Awards of the University's College of Medicine in a program there recently.

* * *

Dr. Merle Musselman, chief of the Department of Surgery at the University of Nebraska, was a recent speaker at a meeting of the Southwest Minnesota Medical Society.

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" NEXT HOMBRE."

Dr. Marie K. Bepko, Cloquet physician, attended the Sixth Pan American Medical Women's Alliance in Miami, Florida. This meeting of women doctors from North, South and Central America and the Caribbean area seeks from each other mutual improvement and encouragement for their participation in all branches of medical public welfare.

Dr. Bepko, before leaving for Miami, attended a course on "Arthritis and Physical Medicine" at the Continuation Center at the University of Minnesota on April 10, 11 and 12.

* * *

Two physicians who have been associated with the Olmsted Medical Group for the past year were elected as partners at the monthly staff meeting of the group. They are **Dr. Malcolm K. Campbell** and **Dr. William R. Weyhrauch.**

* * *

Dr. Saw Mra Aung, medical superintendent of the General Hospital in Mandalay, Burma, was a visitor at the Mayo Clinic and Mayo Foundation from May 5 to 8. Dr. Aung is a participant in the foreign leaders' program of the exchange service, and will visit medical centers in Philadelphia, New York, Boston, Chicago, Minneapolis, Los Angeles, New Orleans, Washington, D. C., and other cities, in addition to Rochester.

* * *

Dr. Frances E. Schaar attended the first annual meeting of the American Society of Hematology in Atlantic City, April 26 and 27.

* * *

Dr. and Mrs. V. M. Baich, Coleraine, and **Dr. and Mrs. A. V. Grinley**, Grand Rapids, attended the Dallas, Texas, meeting of the American Academy of General Practice the last week in March.

* * *

Drs. Joseph Callan, Richard Payne, D. A. Sher and **J. P. Siegel** commented on films about rheumatic fever at Virginia PTA meetings on March 18.

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(*Name of university and results of clinical tests sent upon written request.)

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At the annual meeting of the Greater Saint Paul Community Chest and Council, Inc., **Dr. D. R. Gillespie** was given the 1958 Honorary Award of the Saint Paul Area Public Health Council for "outstanding services to the Council and the Community in general."

* * *

Correction: Dr. N. E. A. Leppo of Duluth turned over his Saturday office in Meadowlands to **Drs. Ed Jorgenson** and **James Troutman,** effective April 1, but continues his pediatric practice in the Medical Arts Building, Duluth.

* * *

Dr. Earl Hill was the speaker at the Wright County Medical Society meeting held at St. Michael on May 6, 1958. He spoke on "Diabetic Coma."

* * *

Dr. Lee Wattenberg, assistant professor of pathology at the University of Minnesota medical school, was one of seventy-nine doctors helped in pursuing academic careers by awards from Lederle Laboratories Division, American Cyanamid Company.

* * *

Dr. Richard A. DeWall is one of seven Minnesota scientists named to share in a total of $66,500 in fellowships for research in heart and blood vessel diseases. The $66,500 was part of $1,433,147 granted 183 scientists in twenty-nine states under the research support program of the American Heart Association and its affiliates. The Minnesota Heart Association will announce its own research awards at its annual meeting, July 8.

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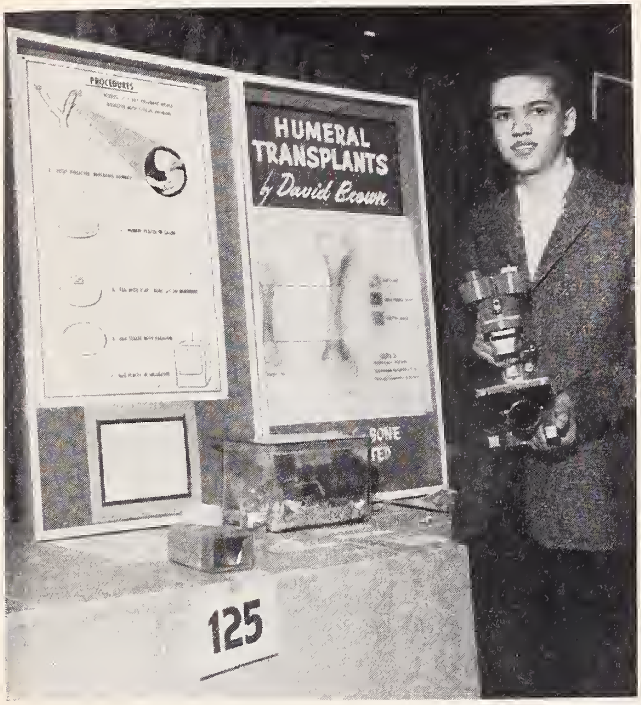
MINNESOTA BOY WINS AMA AWARD

A fifteen-year-old Minneapolis boy will be a guest of the American Medical Association at its 107th Annual Meeting in San Francisco, June 23-27, as a result of winning the Association's top award for the best basic medical science exhibit at the National Science Fair in Flint, Michigan, May 7-10.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Chosen from a field of 281 entries from forty-one states, the District of Columbia, Alaska, Hawaii, Germany, and Japan, David R. Brown, a sophomore at St. Louis Park High School, won the top AMA honor for his exhibit showing how fetal mouse bones become ossified when transplanted into hostile media. A special AMA judging committee, headed by Dr. Alphonse McMahon, St. Louis, chairman of the AMA Council on Scientific Assembly, made the selection and invited David to show his work in San Francisco. His co-winner, sixteen-year-old Clare L. Chatland, of Missoula, Montana, who won for her work with hypersensitized mice, will join him at the annual meeting where they will demonstrate their exhibits to some 30,000 physicians and their guests.

The awards were announced on the final day of the Fair at a special AMA luncheon, attended by 820 students, teachers, regional fair co-sponsors, press and medical representatives. David, who lives at 4011 Excelsior Boulevard, Minneapolis, went to the National Science Fair after winning in the 21st Junior Academy of Science Fair, sponsored by the Minnesota Academy of Science and the Bemidji *Pioneer,* earlier this spring.

* * *

Dr. David Boyd, Jr., professor of psychiatry at the University of Minnesota's Mayo foundation, was the commencement speaker at St. John's University's 101st commencement exercise.

* * *

Dr. James R. Doyle, physician with the Olmsted Medical Group, was elected president of the Rochester Earth Science Society at the annual meeting of that group held in the Rochester High School.

He succeeds Fred C. Kennedy, who held the post two years. Dr. Doyle previously served as vice president.

* * *

Dr. F. G. Gunlaugson, director of the preventable disease division of the Minneapolis health department, is to become tuberculosis control officer for Hennepin county under a contractual arrangement with the Hennepin county sanatorium commission.

The board of public welfare, which has jurisdiction over the health department, approved the proposal. Execution of the contract is now up to the city council. Approval was given previously by state and county authorities.

* * *

Fourteen veteran Duluth physicians were recently honored for service to the medical profession at a Florence Nightingale dinner at Northland Country Club.

Honored were **Drs. William R. Bagley, L. A. Barney, A. N. Collins, W. E. Hatch, C. L. Haney, T. J. Jensen, Harry Klein, A. T. Laird, A. L. McDonald, Mary McCoy, M. A. Nicholson, D. F. Pennie, O. W. Rowe** and **J. A. Winter.**

Sponsors of the event were the St. Louis County Medical society, St. Luke's, St. Mary's and Miller Memorial hospitals, Noepeming sanatorium and the Second District Nurses association.

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Communication

To the Editor:

The recent report of the Ad Hoc Committee of the Surgeon General of the United States, after weighing the advantages and disadvantages of BCG vaccination, states: "It is believed, however, that the advantages of vaccination outweigh the disadvantages for tuberculin negative persons who are exposed to a definite risk of infection, especially if they cannot be retested frequently with tuberculin."

This Committee recommended BCG vaccination for those groups recommended by the American Trudeau Society, the American College of Chest Physicians, the American Medical Association, and the Medical Advisory Committee of Research Foundation.

The specific groups recommended for BCG vaccination by the American Trudeau Society are:

1. Doctors, medical students, and nurses who are exposed to tuberculosis;

2. All hospital and laboratory personnel whose work exposes them to contact with the bacillus of tuberculosis;

3. Individuals who are unavoidably exposed to infectious tuberculosis in the home;

4. Patients and employes in mental hospitals, prisons, and other custodial institutions in whom the incidence of tuberculosis is known to be high;

5. Children and certain adults considered to have inferior resistance and living in communities in which the tuberculosis mortality rate is unusually high.

There have been numerous inquiries from all parts of the United States as to where BCG vaccine may be obtained. In order to assist those interested, the following information is offered:

BCG vaccine may be obtained by any licensed physician in the United States and its territories by writing to the laboratories of Research Foundation and the University of Illinois, the only institutions licensed to produce and distribute BCG in this country. The address of Research Foundation is: 70 West Hubbard Street, Chicago 10, Illinois.

Sol Roy Rosenthal, M.D., Ph.D.
Medical Director, Research Foundation, and
Director, Institution for TB Research,
University of Illinois

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April 21, 1958

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FOR SALE—Westinghouse 100 ma. x-ray unit with most of necessary accessories. About eight years old, and in working order. $500.00. Address E-638, care MINNESOTA MEDICINE.

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FOR SALE—Urology 100 amp x-ray unit, shock-proof cable, late model. Young table, older model. Sacrifice $800.00. Address E-639, MINNESOTA MEDICINE.

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